A Dissertation on

EVALUATION OF THE EFFECT OF BHASTRIKA AND KAPALBHATI PRANAYAMA ON BLOOD GLUCOSE LEVEL IN TYPE 2 DIABETES MELLITUS

Submitted by

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The Institutional Ethics Committee of Government Yoga and Naturopathy Medical College, reviewed and discussed your application for approval of the proposal entitled "EVALUATION OF THE EFFECT OF BHASTRIKA AND KAPALBHATI PRANAYAMA ON BLOOD GLUCOSE LEVEL IN TYPE 2 DIABETES MELLITUS" No. *13052016*

The following members of Ethics Committee were present in the meeting held on 17/05/2016 conducted at Government Yoga and Naturopathy Medical College, Chennai.

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We approve the proposal to be conducted in its presented form

Sd / Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patient's information / informed consent and asks to be provided a copy of the final report.

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LIST OF ABBREVIATIONS USED

2-hr PG	2-hour Plasma Glucose
2-hr PPBG	2-hour Post-Prandial Blood Glucose
ADA	American Diabetes Association
ATP	Adenosine Tri Phosphate
CAM	Complementary & Alternative Medicine
DM	Diabetes Mellitus
ER	Endoplasmic Reticulum
ERV	Expiratory Reserve Volume
FBG	Fasting Blood Glucose
FBS	Fasting Blood Sugar
FEV	Forced Expiratory Volume
FPG	Fasting Plasma Glucose
FVC	Forced Vital Capacity
GK activators	Gluco Kinase Activators
GLP-1	Glucagon-like peptide 1
GLUT4	Insulin-regulated Glucose Transporter
GPCR agonists	G Protein-coupled Receptors agonists
GPR 40 agonists	G Protein-coupled Receptor 40 agonists
GPR21 inhibitors	G Protein-coupled Receptor 21 inhibitors
Group A	Bhastrika Pranayama Group
Group B	Kapalbhati Pranayama Group

GSK-3 inhibitors	Glycogen Synthase Kinase-3 inhibitors
HbA1c	Glycated Hemoglobin Test
HDL	High-Density Lipoprotein
HLA	Human Leukocyte Antigen
HONK	Hyper Osmolar Nonketotic Coma
IAPP	Islet Amyloid Poly Peptide
IAYT	Integrated Approach of Yoga Therapy
IC	Inspiratory Capacity
ICMR	Indian Council of Medical Research
IGF-I & II	Insulin-like Growth Factor I & II
IRS	Insulin Receptor Substrates
IRV	Inspiratory Reserve Volume
LDL	Low-Density Lipoprotein
MAPK	Mitogen-activated Protein Kinases
MEP	Maximum Expiratory Pressure
MS	Metabolic Syndrome
MVV	Maximum Voluntary Ventilation
NCCAM	National Center for Complementary and Alternative Medicine
OGTT	Oral Glucose Tolerance Test
PCOS	Polycystic Ovarian Syndrome
PEFR	Peak Expiratory Flow Rate

PFT	Pulmonary Function Test
PPBS	Post-Prandial Blood Sugar
RCT	Randomized Controlled Trial
ROX	Reactive Oxygen Species
SGLT-2 inhibitors	Sodium-Glucose-Co-Transporter-2 inhibitors
SKY	Sudarshan Kriya Yoga
SVC	Slow Vital Capacity
TENS	Transcutaneous Electrical Nerve Stimulation
Type 2 DM	Type 2 Diabetes Mellitus
UPR	Unfolded Protein Response
VC	Vital Capacity
V_T	Tidal Volume
WHO	World Health Organization

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ABSTRACT

Background: The prevalence of diabetes has been steadily increasing for the past three decades. Many factors including personal, social, and environmental factors influence on the onset and progression of the disease. There is a significant evidence suggest metabolic consequences of stress in an individual suffering from chronic disease like diabetes mellitus. Researches on pranayama, demonstrated its ability to regulate autonomic functions and metabolic consequences brought about by the stress. Previous study on various pranayama in healthy individual has shown improvement in fasting and post-prandial blood glucose level.

Aim: To evaluate and compare the effects of Bhastrika and Kapalbhati Pranayama on blood glucose level in subjects with type 2 diabetes mellitus; A prospective pre-post study design.

Methodology: Fifty subjects aged between 30 – 55 years, pre-diagnosed with Type 2 Diabetes Mellitus, who satisfy inclusion and exclusion criteria were recruited from the Out Patients Department of Government Yoga and Naturopathy Medical College and Hospital, Chennai. They were randomized to two groups. Group A (n=25) practiced Bhastrika Pranayama and Group B (n=25) practiced Kapalbhati Pranayama for five weeks (30 days practice with break of a day after every 6 days). The Fasting Blood Glucose (FBG) and 2-hr Post-Prandial Blood Glucose (PPBG) were estimated on day 1 (baseline data) and day 30 (end-point data). The collected data were properly maintained and statistically analyzed using "*paired't' test"* and "*two-sample't' test*" with Stats 9.0 (College, Station, Texas, USA).

Result: The result demonstrates a statistically significant change in both fasting blood glucose (FBG) and post-prandial blood glucose (PPBG) level in subjects of both Groups (Bhastrika and Kapalbhati group). The overall comparison between the groups revealed that the practice of bhastrika pranyama generated better outcome, compared to those practiced kapalbhati pranayama.

Conclusion: Present study findings suggest that the practice of Bhastrika as well as kapalbhati pranayama found to be effective in improving the biochemical parameters viz. fasting blood glucose and post-prandial blood glucose of type 2 diabetes mellitus. Hence, the practice of pranayama can be implemented in the management of diabetes. Further study of longer duration and large number of samples is necessary to reaffirm the assertion.

Keywords: Type 2 Diabetes Mellitus, Bhastrika Pranayama, Kapalbhati Pranayama, Fasting Blood Glucose, Post-Prandial Blood Glucose.

1.0 INTRODUCTION

Diabetes Mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. **(Kasper et al., 2015)** Chronic hyperglycemic state presents with disturbances of carbohydrate, protein and fat metabolism, occurs either due to inadequate secretion of insulin (a hormone which control blood glucose level and prevents hyperglycemia) or when the body cannot effectively use the insulin produced by beta cells of the pancreatic islets. **(World Health Organization, 1999)**

Diabetes prevalence has been rising more rapidly in middle- and low-income countries. Global prevalence of diabetes among adults over 18 years of age has risen from 4.7% in 1980 to 8.5% in 2014. The number of people with diabetes has risen from 108 million in 1980 to 422 million in 2014. In 2015, an estimated 1.6 million deaths were directly caused by diabetes. Diabetes can be treated and its consequences may be avoided / delayed with proper diet, physical activity, medication, regular screening and treatment of complications. **(World Health Organization, 2016)**

Type 2 diabetes (formerly called non-insulin dependent/adult onset diabetes) arises as a result of body's ineffective use of insulin, almost always with a major contribution from insulin resistance. **(World Health Organization, 1999)** Symptoms of type 2 diabetes mellitus may be similar to that of type 1 diabetes, but are often less marked or absent. Because of this, the disease may go undiagnosed for several years, until complication arises. **(World Health Organization, 2016)**

The pathogenesis of type 2 diabetes is thought to begin with lifestyle-induced insulin resistance, which gradually increases demand on pancreatic beta cells to secrete insulin. **(Swisa et al., 2017)**

Type 2 diabetes mellitus is a chief concern for patients, health care providers and health care systems in America, and around the globe. Individuals with type 2 diabetes mellitus exhibit clinical and subclinical symptoms of anxiety more frequently than people without diabetes. *Anxiety is traditionally associated with poor metabolic outcomes and increased medical complications among those with type 2 diabetes mellitus.* Hence it is proposed link between anxiety and diabetes; offer an innovative and evidence based collaborative care for anxiety and diabetes in primary care. **(Bickett & Tapp, 2016)**

Stress has long been shown to have major effects on metabolic activity. It is a potential contributor to chronic hyperglycemia in diabetes. Although human studies on the role of stress in the onset and course of type 2 diabetes are few, there are more consistent evidence supports the role of stress in type 2 diabetes mellitus. Furthermore, there is mounting evidence of autonomic contributions to the patho-physiology of type 2 diabetes mellitus both in animals and humans. **(Surwit et al., 1992)**

Mood affects food intake: Depression and/or anxiety are risk factors for the development of diabetes **(Bystritsky et al., 2014) (Ljubičić et al., 2013)** and vice versa. **(Guedes et al., 2013) (Ali et al., 2013)** Those patients who had family support or had close relationship with their physician, those who are knowledgeable towards diabetes and those with better confidence in ability to manage self care behaviors had a lower level (better control) of HbA1c. In contrast, patients with depression or stress were significantly correlated with poor glycemic control. Hence, effective and tailored interventions were needed to mitigate exposure to these risk factors, as this would improve better glycemic control and reduce the risks of diabetes complications. **(Badedi et al., 2016)**

Today's clinicians are presented with an extensive range of oral anti-diabetic drugs for type 2 diabetes. Tailoring the treatment to the individual patient is an important principle. Neither sulphonylureas nor biguanides are able to appreciably alter the rate of progression of hyperglycemia in patients with type 2 diabetes. Preliminary data suggesting that thiazolidinediones may provide better long-term glycaemic stability are currently being tested in clinical trials; current evidence, while encouraging, is not conclusive. *However, intensive lifestyle intervention can be more effective than drug therapy*; No anti-diabetic drugs are presently licensed for use in pre-diabetic individuals. **(Krentz & Bailey, 2005)**

Despite, implementation of interventions based on traditional risk factors, the incidence of diabetes continues to rise. Identifying additional factors that contribute to increased risk is of public health significance. It has been estimated that as much as 60% type 2 diabetes risks is due to modifiable environmental factors including obesity, physical inactivity, diet quality, smoking, hypertension and abnormal cholesterol levels. **(Murea et al., 2012 Spring)** It is necessary to look at alternatives, which are not resource-intensive and those which are nearer to the community that people live in. **(Aswathy et al., 2013)**

Yoga holds promise to be a potential therapeutic tool to achieve positive health and cure disease. **(BK, 2007)** *Yoga* aims at bringing the different bodily functions into perfect co-ordination so that they work for the good of the whole body. **(Saraswati, 2009)** Yoga offers a largely unexplored, widely available resource for the management of stress-related ailments. **(Monra et al., 1992)**

Prana (life force) has a very close relation to the mind. As prana of the subtle pranic body (*pranamaya kosha*) is intrinsically linked to the other koshas, *annamaya* (food body), *manomaya* (mental body), etc., prana is influenced by our thoughts, feelings, emotions,etc., and in vice versa they are influenced by prana. **(Saraswati, 2002)**

Breath, the vital force, is controlled positively by **pranayama** to ensure homeostasis and wellbeing in humans. **(Ansari, 2016)** The practice of pranayama clears up the *nadis,* energy pathways in the body. However in an average individual, many of these

pathways are blocked and the chakras release energy only partially. *The negative condition (disease/disorder) we experience, whether physical or mental, are the causes as well as the consequences of the blockages*. With the practice of pranayama, these pathways of energy are gradually freed so that prana moves through them smoothly. **(Saraswati, 2013)**

'*Manu'* – the law giver of the Aryans is of the opinion that pranayama clears the body and mind as the fire cleans dross elements contained in the gold when heated. Almost all the Indian Yogis are of opinion that Asanas free the Sadhaka from Rajoguna; *Pranayama destroys* his *bodily and mental abnormalities if any present*. This shows that Prana and mind are closely related to each other. Mental disturbances like excitement, anxiety, fear, anger, disappointment, lust and other mental aberrations can be calmed down by regular practice of pranayama which results in several health benefits. **(Ramdev, 2004)**

Pranayama consist of three phases: "puraka" (inhalation), "rechaka" (exhalation), and "kumbhaka" (retention); and that can be either fast or slow. Both ***Bhastrika Pranayam*** and ***Kapalbhati pranayam*** belongs to the class of fast breathing technique, which when practiced alone were known to have impact on sympathetic activity and reduce the stress level, from previous studies. **(Sharma et al., 2013)** Though, both pranayama belongs to the fast type of breathing technique they differ in the way that "**bhastrika**" involves both *active inhalation and active exh2eralation* whereas "**kapalbhati**" involves *passive inhalation and active exhalation*.

According to the classic text and research papers, *Bhastrika (bellows breathe)* increases vitality and lowers levels of stress and anxiety by raising the energy and harmonizing the pranas; *Kapalbhati* calms the mind, **(Saraswati, 2008)** tones the digestive organs, balances and strengthens the nervous system, **(Saraswati, 2013)** regulates the neuro-endocrine and autonomic nervous system mechanisms in the body. **(Kekan & Kashalikar, 2013)**

Previous study has shown that the practice of yoga (asanas and pranayama) has made a significant fall in the fasting blood glucose level and one hour postprandial blood glucose level after 40 days of practice, in non-insulin dependent diabetes mellitus persons. **(Malhotra et al., 2004)** In another study, short term intervention of yogic exercises (pranyama) helped in reducing the stress in medical students which is reflected by improvement in both fasting and post meal glucose level. **(Shende et al., 2013)**

Although few studies have shown the practice of pranayama alone improved the blood glucose level in healthy volunteers, no such study has been conducted on diabetic people. Current study was conducted to evaluate and compare the effect of Bhastrika and Kapalbhati Pranayama on blood glucose level in people with Type 2 Diabetes Mellitus.

2.0 AIM AND OBJECTIVE

2.1 AIM

The aim is to study the effect of *Bhastrika and Kapalbhati Pranayama* on blood glucose level in type 2 diabetes mellitus.

2.2 OBJECTIVE

To evaluate and compare the effects of *Bhastrika and Kapalbhati pranayama* on following parameters in type 2 diabetic subjects:

i. Fasting Blood Sugar (FBS)

ii. Post-Prandial Blood Sugar (PPBS)

3.0 REVIEW OF LITERATURE

3.1 DIABETES MELLITUS

Diabetes Mellitus is of three main types namely, ***Type 1 Diabetes Mellitus*** (an autoimmune disease), ***Type 2 Diabetes Mellitus*** (due to improper function or inadequate secretion of insulin) and ***Gestational Diabetes*** (develops during pregnancy). The symptoms are almost similar in both type 1 and type 2 diabetes mellitus, which may develop rapidly (in a weeks or months) in type 1 diabetes, while it usually develops much slowly or may be subtle or sometimes may be absent in type 2 diabetes mellitus. So, the disease may go undiagnosed for several years, until complications arise out of it.

Diabetes is on rise; no longer a disease of predominantly rich nations, rather the prevalence is steadily increasing everywhere, most markedly in the world's low and middle-income countries. **(World Health Organization, 2016)** Type 2 diabetes is the most common type of diabetes, usually occurs in adults, but is increasingly seen in children and adolescents. Although the exact causes for the development of type 2 diabetes are still not known, there are several important risk factors includes excess body weight, physical inactivity and poor nutrition. **(International Diabetes Federation, 2015)** Severe and sustained emotional stress creates a physiological burden through increased sympathetic activity and higher energy demand. This, may lead to increased oxidative stress and lead to the development of metabolic syndrome. *Emotional stress* has been

shown to *contribute to the onset, progression, and control of type 2 diabetes.* Stress management and biofeedback assisted relaxation have been shown to improve glycemic control. **(Munster-Segev et al., 2017)**

3.1.1 INCIDENCE & PREVALENCE

The prevalence of diabetes has been steadily increasing for the past three decades; mirroring an increase in prevalence of overweight and obesity. The number has nearly quadrupled since 1980. **(World Health Organization, 2016)** India leads the world with largest number of diabetics, earning the dubious distinction of being termed "diabetes capital of the world". **(Mohan et al., 2007)** India is second to china which is home for more number of diabetics in the world. **(Gupta et al., 2015)**

South Asian people originating from Pakistan, Bangladesh or India, experience 50% higher risk of developing type 2 diabetes compared with other populations, irrespective of whether they live in South-Asia or Western countries. **(Bhurji et al., 2016)**

It has raised from 108 million in 1980 to 422 million people with diabetes in the year 2014. The global prevalence of diabetes, among the adults over 18 years of age has risen from 4.7% in 1980 to 8.5% in 2014. In the year 2015, an estimated 1.6 million deaths were directly caused by diabetes. WHO projects

that, diabetes will be the 7th leading cause of death by the year 2030. **(World Health Organization, 2017)**

In South East Asia region, it was estimated that about 78.3 million people aged between 20-79 years were affected with diabetes in the year 2015 and the number is projected to raise 140.2 million by the year 2040. **(International Diabetes Federation, 2015)**

Table 3.1 – Global scenario of diabetes

(International Diabetes Federation, 2015)

DIABETES	In 2015	By 2040
Prevalence	One in 11 adults has diabetes	One in 10 adults will have diabetes
Men	215.2 million	328.4 million
Women	199.5 million	313.3 million
Urban area	269.7 million	477.9 million
Rural area	145.1 million	163.9 million

Preliminary results from a large community study conducted by the Indian Council of Medical Research (ICMR) revealed that, lower proportion of the population is affected in the states of Northern India [Chandigarh – 0.12 million, Jharkhand – 0.96 million] as compared to Maharashtra [9.2 million] and Tamil Nadu [4.8 million]. The national urban survey conducted across metropolitan cities of India also reported similar trend which is as follows: Eastern India [Kolkata – 11.7%], Northern India [Kashmir Valley – 6.1%, New Delhi – 11.6%], West India [Mumbai – 9.3%], South India [Chennai – 13.5%, Hyderabad – 16.6%, Bangalore – 12.4%]. **(Kaveeshwar & Cornwall, 2014)**

Genetic predisposition combined with lifestyle associated with urbanization and globalization contributes to the rapid rise of diabetes burden in India. Moreover type 2 diabetes mellitus appears to occur at least a decade earlier in Indian than in European population. According to American Diabetes Association criteria, the prevalence of diabetes was 4.7% in urban and 1.9% in rural areas whereas the prevalence according to World Health Organization criteria was 5.9% in urban and 2.7% in rural areas. The economic burden due to diabetes in India is among the highest in the world. However, the real burden of diabetes is due to its micro-vascular and macro-vascular complication, which leads to increased mortality and morbidity. **(Joshi, 2015)**

3.1.2 CHARACTERISTIC FEATURES OF TYPE 2 DIABETES MELLITUS

Table 3.2 – Characteristic features of type 2 diabetes mellitus, **(Campbell, 2000)**

- Onset usually occurs in patients >30 years of age
- Onset is often insidious
- Positive family history in 30% of cases
- No association with HLA; no islet cell antibodies (as in type 1 diabetes mellitus)
- Symptoms were controlled by the diet and/ or oral hypoglycemic agents, insulin treatments may be required later in the disease if uncontrolled.

3.1.3 CLINICAL PRESENTATION OF TYPE 2 DIABETES MELLITUS

Type 2 Diabetes may present to the clinician in one of the four ways, (i) *classical symptoms* (may be seen at diagnosis) are thirst, polyuria, fatigue and malaise, infections (especially genital candidiasis) and blurred vision (ii) *Incidental diagnosis* include opportunistic urine and blood testing for glucose done in older individuals at the time of illness, either at the primary or secondary care level, or as a part of an annual health check (iii) *Complications of diabetes* viz. micro- and macro-vascular disorder (iv) *Hyperosmolar Nonketotic Coma (HONK)* is a form of diabetic coma seen in older type 2 diabetic patients often seen at diagnosis in previously undiagnosed subjects. **(Campbell, 2000)**

3.1.4 DIAGNOSTIC CRITERIA

Table 3.3 – Criteria for the diagnosis of diabetes

(American Diabetes Association, 2017)

Fasting Plasma Glucose (**FPG**) ≥ 126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hr.*
OR
2-hour Plasma Glucose (**2-h PG**) ≥ 200 mg/dL (11.1 mmol/L) during an **Oral Glucose Tolerance Test** (**OGTT**). The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in water.*
OR
HbA1C ≥ 6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is NGSP[1] certified and standardized to the DCCT[2] assay.*
OR
In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥ 200 mg/dL (11.1 mmol/L).
*In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing.

[1] – National Glycohemoglobin Standardization Program

[2] – Diabetes Control and Complications Trial

Table 3.4 – Oral Glucose Tolerance Test (OGTT)

(Frier & Fisher, 2010)

How to perform an Oral Glucose Tolerance Test (OGTT)		
Preparation before the test		
• Unrestricted carbohydrate diet for 3 days • Fasted overnight for at least 8 hrs • Rest for 30 mins • Remain seated for the duration of the test, with no smoking		
Sampling		
• Plasma glucose is measured before and 2hrs after a 75 g oral glucose drink		
Interpretation (venous plasma glucose)		
	Fasting	**2hrs after glucose load**
Fasting hyperglycemia	6.1-6.9 mmol/L (110-125 mg/dL)	< 7.8 mmol/L (< 140 mg/dL)
Impaired glucose tolerance	< 7.0 mmol/L (< 126 mg/dL)	7.8-11.0 mmol/L (140-199 mg/dL)
Diabetes	≥ 7.0 mmol/L (≥ 126 mg/dL)	≥ 11.1 mmol/L (≥ 200 mg/dL)

Indication for diabetes screening in asymptomatic individuals includes the following criteria: **(Khardori, 2017)**

- Sustained blood pressure of >135/80 mmHg
- Overweight and one or more other risk factors for diabetes (e.g., blood pressure > 140/90 mmHg, HDL level < 35 mg/dL, triglyceride level > 250 mg/dL)
- ADA recommends screening at age of 45 years in the absence of the above criteria.

3.1.5 RISK FACTORS

Though researchers fully don't understand why some people develop type 2 diabetes and others do not, however it is clear that certain factors increases the risk of developing type 2 diabetes which includes weight, fat distribution, physical inactivity, family history, race, age, pre-diabetes, gestational diabetes and polycystic ovarian syndrome. **(Mayo Clinic, 2016)**

i. **Weight** – Overweight being the primary risk factor to develop diabetes, though people need not to be overweight to develop diabetes. If more fatty tissue present in a body, more resistant the cells become to insulin.

ii. **Fat distribution** – If there is more fat deposition in or around the abdomen, greater is the risk to develop type 2 diabetes compared with that of fat deposition elsewhere (hips/thighs) in the body.

iii. **Inactivity** – Physical activity helps to control weight by using glucose as energy and makes cells more sensitive to insulin. Physical inactivity or less activity increases the risk of type 2 diabetes.

iv. **Family history** – Greater is the risk to develop diabetes, if parents/siblings has type 2 diabetes.

v. **Race** – Although it is not clear why certain race have greater incidence, certain races like blacks, Hispanics, American Indians and Asian Americans are more likely to develop type 2 diabetes compared with that of whites.

vi. **Age** – Older the individual more risk is to develop type 2 diabetes due to less exercise, lose muscle mass and weight gain, especially after the age of 45. But more recently, there is dramatic increase in the incidence of type 2 diabetes even among children, adolescent and younger adults.

vii. **Prediabetes** – Is a condition in which blood sugar level is higher than normal, but not high enough to be classified as diabetes, often may progresses to type 2 diabetes.

viii. **Gestational diabetes** – If a woman gets diabetes during pregnancy or is she gives birth to baby weighing more than 9 pounds (4 Kilogram), risk of developing type 2 diabetes increases.

ix. **Polycystic ovarian syndrome** – Women having PCOS – a condition characterized by irregular menstrual cycle, excess bodily hair growth and obesity, increases the risk of diabetes.

3.1.6 COMPLICATIONS

Table 3.5 - Complications of diabetes mellitus

(Graham Douglas, 2011)

Micro vascular / neuropathic complications
Retinopathy – cataract, impaired vision Nephropathy – protein loss, renal failure Peripheral neuropathy – sensory loss, motor weakness Autonomic neuropathy – postural hypotension, vomiting, diarrhea Foot disease – ulceration, arthropathy
Macro vascular complications
Coronary circulation – Myocardial ischaemia/infarction Cerebral circulation – Transient ischaemic attack (TIA), stroke Peripheral circulation – Claudication, gangrene, amputation

Other possible complication includes the following: **(Mayo Clinic, 2016)**

- Hearing impairment – Hearing problems are more common in people with diabetes
- Skin Conditions – Diabetes makes more susceptible to skin problems, includes bacterial & fungal infections.
- Alzheimer's disease – Type 2 DM may increase the risk of Alzheimer's. Poorer the blood control greater is the risk, but the exact connection between the 2 still remains unclear

3.1.7 PATHOPHYSIOLOGY (STRESS MECHANISM)

Type 2 diabetes mellitus is characterized by combination of peripheral insulin resistance and inadequate secretion of insulin by the pancreatic beta cells. Insulin resistance which has been attributed to elevated levels of free fatty acids & pro-inflammatory cytokines in the plasma, that leads to decreased glucose transport into the muscle cells, elevated hepatic glucose production and increased breakdown of fat from body tissue. **(Khardori, 2017)** Pathophysiology of Type 2 diabetes mellitus and its co-morbidities has been correlated with *stress mechanism*. Stress suppresses body's immune system and neuro-humoral actions thereby affecting the normal psychological state. **(Singh et al., 2015)**

The hyperglycemic effects of the stress were noted as early as in the 17th century by *Thomas Willis;* His work was followed in 1849 by *Claude Bernard*, who demonstrated that *"causing lesion in an area of hypothalamus in normal rabbits causes hyperglycemia",* giving a early credence to theories that *hypothalamic pituitary axis plays a distinct role in the development of hyperglycemia.* In 1930, *C.F. Cori,* who chose '*pharmacological stress response mechanism'* for his experiment with rabbits and theorized the link between the *physiological stress response and development of hyperglycemia.* Later *Cannon* examined the '*response to a physiological stress'* in cats and found that the *glycosuria was absent at baseline, but developed in animals that*

were observed to respond to restraint with emotions of fright or rage. **(Surwit, 2008)**

The potential role of stress in type 2 diabetes mellitus was noted a century ago by *William Osler* and recommended stress-reducing maneuvers for the treatment for this form of disease. More recently a theoretical rationale for the importance of stress effects, on glycemic control in type 2 diabetes mellitus has emerged from the animal studies. These uniformly suggest that *stress can affect glycemic control adversely in type 2 diabetes mellitus.* Evidence from both animal and human studies suggests that *individuals with type 2 diabetes have altered adrenergic sensitivity in the pancreas,* and perhaps other site as well, which could make the individual particularly sensitive to stressful environmental stimuli; other stimuli of sympathetic activity such as dietary fat and simple carbohydrates also may contribute more to the development of diabetes mellitus through this adrenergic mechanism. **(Surwit et al., 1992)**

3.1.7.1 How does stress increase blood glucose?

Whenever there is a stress, the glucose needs to go up. This rise is essential to supply the extra energy that is necessary for the highly increased muscle work to run away from the sensed danger. *The endocrine and autonomic nervous system that regulate all the stress adaptation mechanism are also involved in the regulation of the blood glucose level*. In addition to this, there is a separate direct regulation of the blood glucose regulating hormonal system that is active

not only during stress but it monitors and regulates a constant level of glucose in the blood all the time. The hormone that increases the blood glucose level is *"glucagon"* and that which reduces is *"insulin"*. Whenever there is a threat or danger, the sympathetic nerves system activates and release large qualities of stress chemicals called *adrenalin* and *nor-adrenalin*. Stress also releases other hormones such as *cortisol (steroids), thyroid hormones, and growth hormones* in to the blood. All these chemicals actively increases the blood glucose levels and thus during stress the blood glucose may shoot up much beyond the levels necessary. Although, this process of excessive release of blood glucose during emotional stress is common to both diabetics and non-diabetics, it remains high in diabetics because necessary amount of the insulin from the target beta cells is not released instantaneously to transport all excess glucose into the cells. Because of the inability (*insufficiency or inefficiency*) of the cell to manage to supply this sudden demand, the blood glucose remains high for a long time even after a short duration of emotional upsurge. Although, the external situations are not posing demand, on the emotional state of the individual the internal restlessness, unquenchable ambitions, unnecessary anxieties and apprehensions of unfounded fears, could be the major demands that can keep the blood sugar at very high levels. Repeated long standing emotionally demanding situations with large quantities of stress tend to increase blood glucose level even if we are more conscious with food and exercise. Although it is unclear whether psychosocial stress exerts a direct psychosomatic effect on the neuro-endocrine regulatory mechanisms that influence metabolic control, it

is quite clear that stress can influence a person's compliance behavior in ways that lead to difficulties in metabolic control. **(Srikanta et al., 2010)**

The pancreatic beta cell failure was categorized as follows. (i) ***Reduced Beta Cell Numbers*** – beta cell death resulting in reduced beta cell mass that cannot deliver sufficient insulin or alternatively it is possible, if the individual with a lower potential for beta cell regeneration fail to increase beta cell mass when demand is higher, opening a gap between demand and supply, which eventually causes hyperglycemia. (ii) ***Beta Cell Dysfunction*** – it has been proposed that *metabolic load may lead to severe endoplasmic reticulum (ER) stress in beta cells*, and that cellular adaptation to ER stress namely the unfolded protein response (UPR), can reduce the glucose stimulated insulin secretion. Otherwise chronic oxidative stress resulting from improper glucose metabolism may cause pancreatic beta cell dysfunction. (iii) ***Loss of Beta Cell Identity*** – as a capable cell for synthesizing, processing and secreting mature insulin in response to metabolic, hormonal and neurologic stimuli or at the molecular level cell that express the full complement of genes associated with secretion of regulated normal insulin, if it loses its identity may eventually lead to type 2 diabetes. **(Swisa et al., 2017)**

3.1.7.2 Regulation of Glucose Homeostasis

3.1.7.2 (a) Overall regulation of glucose homeostasis

Glucose homeostasis reflects a balance between hepatic glucose production and peripheral glucose uptake and utilization. Insulin is the most important regulator of this metabolic equilibrium, but neural input, metabolic signals, and other hormones (e.g., glucagon) result in integrated control of glucose supply and utilization. The organs that regulate glucose and lipids communicate by neural and humoral mechanisms with fat and muscle producing adipokines, myokines, and metabolites that influence liver function. In the fasting state, low insulin levels increase glucose production by promoting hepatic gluconeogenesis and glycogenolysis and reduce glucose uptake in insulin-sensitive tissues (skeletal muscle and fat), thereby promoting mobilization of stored precursors such as amino acids and free fatty acids (lipolysis). Glucagon, secreted by pancreatic alpha cells when blood glucose or insulin levels are low, stimulates glycogenolysis and gluconeogenesis by the liver and renal medulla. Postprandially, the glucose load elicits a rise in insulin and fall in glucagon, leading to a reversal of these processes. *Insulin, an anabolic hormone, promotes the storage of carbohydrate and fat and protein synthesis*. The major portion of postprandial glucose is used by skeletal muscle, an effect of insulin-stimulated glucose uptake. Other tissues, most notably the brain, use glucose in an insulin-independent fashion. Factors secreted by skeletal myocytes (irisin), adipocytes (leptin, resistin, adiponectin, etc.), and bone also influence glucose homeostasis. **(Powers, 2015)**

3.1.7.2 (b) Insulin Biosynthesis

Insulin is produced in the beta cells of the pancreatic islets. It is initially synthesized as a single-chain 86-amino-acid precursor polypeptide, preproinsulin. Subsequent proteolytic processing removes the amino-terminal signal peptide, giving rise to proinsulin. Proinsulin is structurally related to insulin-like growth factors I and II, which bind weakly to the insulin receptor. Cleavage of an internal 31-residue fragment from proinsulin generates the C peptide and the A (21 amino acids) and B (30 amino acids) chains of insulin, which are connected by disulfide bonds. The mature insulin molecule and C peptide are stored together and co-secreted from secretory granules in the beta cells. Because C peptide is cleared more slowly than insulin, it is a useful marker of insulin secretion and allows discrimination of endogenous and exogenous sources of insulin in the evaluation of hypoglycemia. Pancreatic beta cells co-secrete islet amyloid polypeptide (IAPP) or amylin, a 37-amino-acid peptide, along with insulin. The role of IAPP in normal physiology is incompletely defined, but it is the major component of the amyloid fibrils found in the islets of patients with type 2 diabetes, and an analogue is sometimes used in treating type 1 and type 2 DM. Human insulin is produced by recombinant DNA technology; structural alterations at one or more amino acid residues modify its physical and pharmacologic characteristics **(Powers, 2015)**

3.1.7.2 (c) Insulin Secretion

Glucose is the key regulator of insulin secretion by the pancreatic beta cell, although amino acids, ketones, various nutrients, gastrointestinal peptides, and neurotransmitters also influence insulin secretion. Glucose levels >3.9 mmol/L (70 mg/dL) stimulate insulin synthesis, primarily by enhancing protein translation and processing. Glucose stimulation of insulin secretion begins with its transport into the beta cell by a facilitative glucose transporter. Glucose phosphorylation by glucokinase is the rate-limiting step that controls glucose-regulated insulin secretion. Further metabolism of glucose-6-phosphate via glycolysis generates ATP, which inhibits the activity of an ATP-sensitive K+ channel. This channel consists of two separate proteins: one is the binding site for certain oral hypoglycemic (e.g., sulfonylureas, meglitinides); the other is an inwardly rectifying K+ channel protein (Kir6.2). Inhibition of this K+ channel induces beta cell membrane depolarization, which opens voltage-dependent calcium channels (leading to an influx of calcium) and stimulates insulin secretion. Insulin secretory profiles reveal a pulsatile pattern of hormone release, with small secretory bursts occurring about every 10 min, superimposed upon greater amplitude oscillations of about 80–150 min. Incretins are released from neuroendocrine cells of the gastrointestinal tract following food ingestion and amplify glucose-stimulated insulin secretion and suppress glucagon secretion. Glucagon-like peptide 1 (GLP-1), the most potent incretin, is released from L cells in the small intestine and stimulates insulin secretion only when the blood glucose is above the fasting level. Incretin

analogues or pharmacologic agents that prolong the activity of endogenous GLP-1 enhance insulin secretion. **(Powers, 2015)**

3.1.7.2 (d) Insulin Action

Once insulin is secreted into the portal venous system, ~50% is removed and degraded by the liver. Un-extracted insulin enters the systemic circulation where it binds to receptors in target sites. Insulin binding to its receptor stimulates intrinsic tyrosine kinase activity, leading to receptor autophosphorylation and the recruitment of intracellular signaling molecules, such as insulin receptor substrates (IRS). IRS and other adaptor proteins initiate a complex cascade of phosphorylation and dephosphorylation reactions, resulting in the widespread metabolic and mitogenic effects of insulin. As an example, activation of the phosphatidylinositol-3′-kinase (PI-3-kinase) pathway stimulates translocation of a facilitative glucose transporter (e.g., GLUT4) to the cell surface, an event that is crucial for glucose uptake by skeletal muscle and fat. Activation of other insulin receptor signaling pathways induces glycogen synthesis, protein synthesis, lipogenesis, and regulation of various genes in insulin-responsive cells. **(Powers, 2015)**

3.1.7.3 How Insulin Resistance develops?

Insulin resistance, a complicated condition in which three primary metabolic tissues viz. skeletal muscle, liver and white adipose tissue sensitive to insulin become less sensitive and its downstream metabolic actions under normal

serum glucose concentration. *Inflammation plays an important role in the development of insulin resistance via various cytokines and molecular pathways.* **(Chen et al., 2015)**

While genetics, aging and ethnicity plays a major role in developing insulin sensitivity, the driving force behind the development of insulin resistance include, excess body weight, increased fat deposition in abdomen, lack of exercise, smoking and skimping on sleep. As insulin resistance develops, the body tries to fight back by producing more amount of insulin. Over the months and years, β-cells of pancreas that works so hard to make insulin gets worn out and can no longer keep pace with the demand for insulin requirement. After years of insulin resistance, blood sugar may begin to rise which may consequently lead to development of prediabetes or type 2 diabetes. **(Harrar, 2016)**

Table 3.6 - Illustrate some causes of Insulin Resistance

(Guyton & Hall, 2006)

Some causes of Insulin Resistance
• Obesity/overweight (especially excess visceral adiposity) • Excess glucocorticoids (Cushing's syndrome or steroid therapy) • Excess growth hormone (acromegaly) • Pregnancy, gestational diabetes • Polycystic ovary disease • Lipodystrophy (acquired or genetic; associated with lipid accumulation in liver) • Autoantibodies to the insulin receptors • Mutations of insulin receptors • Mutations of the peroxisome proliferators' activator receptor γ (PPARγ) • Mutation that cause genetic obesity (e.g., melanocortin receptor mutations) • Hemochromatosis (a hereditary disease that causes tissue iron accumulation)

3.1.7.4 Type 2 Diabetes Mellitus: Direct and Indirect impact of stress

At present, the causes of Type 2 Diabetes are not entirely clear, but predictors have been found in recent studies namely, obesity, hypertension, and sedentary lifestyle, alterations in the glycemic status and lipid metabolism correlate with Type 2 Diabetes and its diffusion. Insulin resistance and other conditions with degrees of glucose intolerance commonly occur together with a collection of clinical and biochemical features, known as "*Metabolic Syndrome*" (MS). The term defines a cluster of components that reflect over-nutrition, sedentary lifestyles and an excess of adiposity. Metabolic Syndrome is usually a pre-clinical condition; genetic predisposition and lifestyle (overweight, sedentary lifestyle, bad dietary habits) lead individuals to develop Type 2 Diabetes Mellitus. *The physiological changes triggered by stress may directly affect the endocrine and immune system. On other hand, the reaction to the stressor (in some cases) may consist, developing an unhealthy lifestyle, neglect of physical well-being, eating in a disorderly fashion, often using food in a consoling or compensatory manner may indirectly affect the risk of developing the disease - Type 2 Diabetes Mellitus*. **(Falco et al., 2015)**

3.1.7.4 (i) Stress – Cortisol – Diabetes

Cortisol is one of the prime actors mediating the effect of stress on metabolism in general and on glucose metabolism in specific. It increases the blood glucose levels by *stimulating hepatic gluconeogenesis and inhibiting the action of insulin.* This reaction is entirely useful in case of fight and flight reaction but

are not entirely suited to cope with the stressors triggered by modern life, which are mostly relational, intangible and durable that may induce chronic hypercortisolism, likely to facilitate the onset of metabolic syndrome and Type 2 Diabetes Mellitus. **(Falco et al., 2015)**

3.1.7.4 (ii) Disease itself as a source of stress

Getting ill may cause personal and interpersonal conflicts, where normal rhythms of the life are disrupted, forcing an individual to question personal values and long-term objectives. Type 2 Diabetes require a complex and largely self-managed treatment which includes daily use of drugs (insulin/hypoglycemic agents), regular measurement of blood glucose levels, special attention to diet and everyday activities. The requirement of shift from the established routines may create emotional and cognitive fatigue. The need to control aspects of life which were once considered to be very normal can be experienced as loss of freedom and spontaneity. Some people may develop an image that their body is different from the past and compared to that of the peers, this diversity might be interpreted in a negative way. On behavior aspect, one can observe different reactions, depending not only on severity of clinical situation, but also based on his/her personality, self-esteem and social support. Hence, the early identification and treatment of these issues may help to develop an adaptive style of coping, which will bring about positive result on compliance and metabolic balance. In addition, it may help to prevent the risk of long term complications. **(Falco et al., 2015)**

3.1.7.4 (iii) Impact of stress on diabetes progression

It is difficult to define or measure the role of stress in the etiology of diabetes, but there is significant evidence suggest of stress's' metabolic consequences in an individual who already suffering from chronic disease like diabetes mellitus. Psychological strain activates the neuro-endocrine mechanism, which influence the blood glucose level through the release of cortisol, endorphins and growth hormones. This mechanism has an adaptive importance in a healthy individual whereas the chronic stress-induced hyperglycemia may aggravate the disease. These mechanisms usually affects the young people, for whom the endocrine system continuously undergoes adaptation making them particularly sensitive to the effects of environmental stimuli whereas in case of the elderly people, *stress plays a major role in the development of complications* such as neuropathy, nephropathy and retinopathy. Along with this, if a person presents with *negative emotions*, *may reduce and undermine the willingness to comply with the treatments and diets*; thus a vicious cycle of poor compliance, poor glycemic control, nervousness and physical vulnerability is established. This, in turn makes the people more difficult to adapt to cope with any new problem that arises. Therefore, care must be given to the people suffers with diabetes, through the process which supports resilience and improving the self-efficacy of the person, favoring the activation of problem-focused coping in them. *Treatment plans should also involve the families so they can help their family member to adapt to the illness.* Some *research in the developmental psychology* has shown that *'there is a connection between parents' stress and that of their*

diabetic children' which may influence the quality of glycemic control. Hence, family support is also an important factor in adult diabetic patient. Partner's attitude influences the adherence to the treatment and the psychological impact on the disease has been shown to be more serious in type 2 diabetics who live alone compared to type 2 diabetics who were with their family support. **(Falco et al., 2015)**

3.1.8 CONVENTIONAL MANAGEMENT

The goals of the therapy for type 2 diabetes mellitus are to (i) eliminate the symptoms related to hyperglycemia, (ii) reduce or eliminate the long-term microvascular and macrovascular complications of diabetes and (iii) allow the patient to achieve as normal a lifestyle as possible. To reach these goals, the physicians should identify target level of glycemic control for each patient; provide the patient with educational and pharmacological resources necessary to reach this level, and monitor/treat diabetes related complications. **(Powers, 2015)**

3.1.8.1 PHARMACOTHERAPY

The main classes of the drugs used in the management of type 2 diabetes are heterogeneous in their modes of action, safety profiles and tolerability which include agents that stimulate insulin secretion *(sulphonylureas and rapid-acting secretagogues)*, reduce hepatic glucose production *(biguanides)*, delay digestion and absorption of intestinal carbohydrate *(alpha-glucosidase*

inhibitors) or improve insulin action *(thiazolidinediones)*. **(Krentz & Bailey, 2005)** The new drug classes have indeed come in the market such as *SGLT-2 inhibitors* and others are in the experimental stages such as *GPR 40 agonists, GSK-3 inhibitors, GK activators* and *GPR21 inhibitors* which definitely could be anticipated as safe and effective, for diabetes therapy. **(Kumar et al., 2017)**

3.1.8.2 SURGICAL INTERVENTION

Bariatric surgery is emerging as a powerful weapon against obesity and type 2 diabetes. There is accumulating evidence that surgery with gastrointestinal manipulations has a role in metabolic regulation and may result in remission of type 2 diabetes (metabolic surgery). The major mechanisms mediating the weight loss-independent effects of bariatric surgery comprise, its effects on *tissue-specific insulin sensitivity, β-cell function & incretin responses, change in bile acid composition & flow, modification of gut flora, intestinal glucose metabolism and increased brown adipose tissue metabolic activity.* According to recently released guidelines, *bariatric surgery* should be *recommended in diabetic patient wh*o satisfies one or more of the following criteria: (i) *class III obesity, regardless of their level of glycemic control*; (ii) *class II obesity with inadequately controlled type 2 diabetes despite change in lifestyle and optimal medical therapy;* (iii) *class I obesity and inadequately controlled hyperglycemia, despite optimal medical treatment.* **(Koliaki et al., 2017)**

3.1.9 YOGA AND TYPE 2 DIABETES MELLITUS

The concept of disease is found in the treatise called *"**Yogavāsiṣṭha**"*. According to Yogavāsiṣṭha, the modern diseases such as asthma, diabetes, hypertension, and anxiety are called *"**Ādhija Vyādhi**" **(stress born diseases)***. These stress born diseases originating in *Manomaya Kośa* – the astral layer of our existence. They arise from our actions that are governed by our emotions [strong likes and dislikes] rather than what is right or wrong. Often in this phase, we respond to emotions – the pull of senses knowing completely well that we are going against what is right. This is called *Prajnāparādha* in Āyurveda – mistake at the level of inner consciousness. It is this '*going against what is right'* (the cosmic law) that causes imbalance – a disease at the Manomaya Kośa called Ādhi. If it is not approached properly, this will bring imbalances at the level of Prāṇamaya Kośa that shows up as *breathing jerks, imbalances and speed.* This in turn creates *stress reactions* causing autonomic and endocrine imbalances leading to diseases in the body called "***Vyādhi***". Yoga practices help in bringing about the balance at all the level of existence (five Kośas) thereby, complete health can be restored. **(Srikanta et al., 2010)** Yoga is a mind-body technique being holistic in nature; it is the best means for achieving physical, mental, social and spiritual well being in an individual. **(Madanmohan, n.d.)** To advocate yoga as a cost-effective primary treatment especially for people with mild-moderate type 2 diabetes, counseling at individual/family level and awareness at community level need to be planned. **(More & Jagannathan, 2015)**

3.1.10 MECHANISM OF ACTION OF YOGA IN DIABETES MELLITUS

A proposed model of mechanism of yoga in people with diabetes mellitus involves two interactive pathways, (i) *by reducing the activation and reactivation of the sympathoadrenal system and the hypothalamic pituitary adrenal axis and promoting feelings of well-being.* Yoga may alleviate the effects of stress and encourage multiple positive downstream effects on metabolic function, neuro-endocrine status and related systemic inflammatory responses (ii) *by directly stimulating the vagus nerve,* yoga may enhance parasympathetic activity and lead to positive changes in cardiovagal function, in mood and energy state, in related neuro-endocrine, metabolic and inflammatory responses. In many research studies, yogic intervention has been show to positively influence several risk factors of diabetes mellitus such as stress, insulin resistance, obesity and sedentary life style. **(Cramer et al., 2015)** Pranayama breathing exercises appear to alter autonomic responses to breath holding probably by increasing the vagal tone and decreasing sympathetic discharges. **(Bhargava et al., 1988)**

3.2 RESEARCH ARTICLE - REVIEW

3.2.1 PSYCHOSOCIAL STRESS AND TYPE 2 DM

Stress has long been suspected as having major effects on metabolic activity. The effect of stress on glucose metabolism are mediated by a variety of "counter-regulatory" hormones that are released in response to stress and that result in elevated blood glucose levels and decreased insulin action. This energy mobilizing effect is of adaptive importance in a healthy organism. However, in diabetes, because of a relative or absolute lack of insulin, stress-induced increases in blood glucose cannot be adequately metabolized. Thus, stress is a potential contributor to chronic hyperglycemia in diabetes, although its exact role is unclear. They concluded that further research is needed to establish the importance of behavioral factors in the etiology and management of diabetes, and several areas of methodological improvement are suggested. **(Surwit & Schneider, 1993)**

Many patients believe that their diabetes has been caused by stress or an adverse life event. Whereas there is strong evidence that psychological stress is related to a deterioration in glycemic control in established diabetes, there is much less evidence that psychological stress can cause diabetes in humans de novo. **(Wales, 1995)**

Herpertz *et al.,* 2000 found that a considerable number of diabetic patients suffer from extreme psychosocial stress often associated with poor diabetic

control. They concluded that, these patients need psychosocial care which should primarily be offered in diabetologic centers incorporating both the patients' family and family background. **(Herpertz et al., 2000)**

3.2.2 OXIDATIVE STRESS AND TYPE 2 DM

Oxidative stress – a deleterious factor which is observed to be increased in metabolic syndrome, leading to dyslipidaemia, β-cell dysfunction. Impaired β-cell functioning results in an under production of insulin, impairs glucose stimulated insulin secretion, fasting hyperglycemia and eventually the development of Type 2 Diabetes Mellitus. **(Tangvarasittichai, 2015)**

Oxidative stress is produced under diabetic conditions and is likely involved in progression of pancreatic beta-cell dysfunction. Possibly caused by low levels of antioxidant enzyme expression, pancreatic beta-cells are vulnerable to oxidative stress. When beta-cell-derived HIT-T15 cells or isolated rat islets were exposed to oxidative stress, insulin gene expression was markedly decreased. In their study, they evaluated the effects of antioxidants in diabetic C57BL/KsJ-db/db mice. From their observation they concluded that, oxidative stress and consequent activation of the *JNK pathway (Jun amino-terminal kinases/c-Jun N-terminal Kinase are members of the MAPK-Mitogen Activated Protein Kinase family and are activated by a variety of environmental stresses, inflammatory cytokines, growth factors and GPCR-G Protein-Coupled Receptors agonists)* are involved in progression of beta-cell dysfunction found

in diabetes. Antioxidants may serve as a novel mechanism-based therapy for type 2 diabetes. **(Kajimoto & Kaneto, 2004)**

Robertson *et al.,* 2007 made a hypothesis that residual hyperglycemia, especially after meals generates reactive oxygen species (ROS), which in turn causes chronic oxidative stress on the beta cell. The molecular mechanism responsible for the glucose toxic effect on beta cell function involves disappearance of two important regulators of insulin promoter activity, PDX-1 (*also known as insulin promoter factor 1*) and MafA. Antioxidant treatment in vitro prevents disappearance of these two transcription factors and normalizes insulin gene expression. These observations suggest that the ancillary treatment with antioxidants may improve outcomes of standard therapy of type 2 diabetes in humans. **(Robertson et al., 2007)**

3.2.3 COMPLEMENTARY & ALTERNATIVE MEDICINE AND TYPE 2 DM

Complementary and Alternative Medicine (CAM) is a rapidly evolving field of medicine that consists of therapy used as an adjuvant or alternative to the conventional medicines/therapy. The *National Center for Complementary and Alternative Medicine (NCCAM)* defines CAM as 'a group of diverse medical and health care systems, practices and products that are not presently considered to be part of conventional medicine'. CAM therapies are broadly classified by the *NCCAM* into: (i) *biologically based therapies* (e.g. herbs, dietary supplements, aromatherapy, oxygen therapy), (ii) *mind-body interventions* (e.g. yoga, hypnotherapy), (iii) *energy therapies* (e.g. Reiki, Tai Chi), (iv) *manipulative and body based methods* (e.g. chiropractic, reflexology), and (v) *alternative medical systems* (e.g. homeopathy, Chinese herbal medicine, naturopathy). The CAM include *major therapeutic lifestyle changes which are accessible, effective and cost-effective* when used alone or as an adjuvant, which offer improvements in physical health, self-esteem and quality of life. **(Suri et al., 2016)**

Lee *et al.,* 2004 made a survey to measure the prevalence of complementary and alternative medicine (CAM) use among Korean diabetes mellitus patients. Out of 223 respondents, 65% had taken CAM products, while 35% of them had not taken the CAM products. A total of 57.9% of the users felt that it was effective, 30.3% of the user considered that it regulated their blood glucose level and 27.6% users felt that it was effective in achieving psychological

relaxation. Out of *ninety three CAM materials* used by the respondents, 63.7% were plant based materials, 21.6% were animal materials and 14.7% were the mixture of both. The majority of the diabetes patients (81.2%) were trying new type of CAM materials and one-third of all patients were trying to start using new CAM products after consulting with their physicians. They concluded that doctors should recognize that CAM were widely used by the diabetic patients and should appreciate that these medicine can even cause adverse effects, so they should therefore be prepared to question patients to determine what non-conventional medicines they are using and rule out what possible outcome that may bring about in their patients. **(Lee et al., 2004)**

Ogbera *et al.*, 2010 in their study found that CAM usage is an important facet of management of diabetes mellitus among their patients, with biological based therapies being the most prevalent form of CAM being utilized. Despite complementary & alternative medicine usage, adherence to prescribed medications was high and they concluded that further evaluation of the impact of CAM on hyperglycemia is needed. **(Ogbera et al., 2010)**

Manya *et al.*, 2012 concluded that respondents with diabetes, who participated in the study, frequently use CAM to treat their diabetes and for their general health. Health care professionals should be aware of CAM use by people with diabetes and take a through history to document any such usage by their

patients so as to monitor outcomes which might either be a beneficial or potential side effects produced by CAM usage. **(Manya et al., 2012)**

3.2.4 WATER DRINKING AND TYPE 2 DM

Naumann *et al.,* 2017 made a meta-analysis of prospective cohort studies, other observational studies, studies with animal models and interventional studies using hydrogencarbonated and magnesium supplements, *suggest a probable positive effect of drinking water and mineral water in particular on glycemic parameters.* Supporting positive results were found in some of randomized controlled studies, especially those substituting diet beverages/caloric beverages with water/bicarbonate and magnesium-rich water. They concluded that because of the high prevalence and associated suffering which resulting in more health expenditures, it is imperative to conduct large and more rigorous trials to answer the question whether water drinking/mineral water can improve glycemic parameters in diabetic and non-diabetic individuals. **(Naumann et al., 2017)**

3.2.5 HOT-TUB THERAPY FOR TYPE 2 DM

Philip L. Hooper, 1999 in his study on hot-tub therapy, patients reported improved sleep and an increases sense of well-being during the course of study. It may be especially useful for the patients who are unable to do exercise. The benefits could result from the increased blood flow to the skeletal muscles. The outcome of the study suggests that hot-tub therapy should be further evaluated

as a therapy for patients with type 2 diabetes mellitus. **(Philip L. Hooper, 1999)**

3.2.6 VITAMIN E AND DIABETES MELLITUS

Jain & Jain, 2012 in their study concluded that "Vitamin E supplementation has an important role in delaying the onset of the diabetic complications as well as for slowing down the progression of diabetic complications". **(Jain & Jain, 2012)**

3.2.7 HERBAL ACUPUNCTURE AND TYPE 2 DM

Lee *et al.,* 2017 made a meta-analysis with seven RCTs on herbal acupuncture. The herbal acupuncture found to significantly reduced levels of fasting blood glucose, 2-hour postprandial glucose and HbA1C. However, there was no significant effect of herbal acupuncture on total cholesterol, HDL, LDL or triglyceride level. They concluded that herbal acupuncture might have a beneficial effect on FBG, 2-hr PPBG, and HbA1C levels in patients with Type 2 DM. **(Lee et al., 2017)**

3.2.8 ACUPUNCTURE AND TYPE 2 DM

Peplow, 2015 reported that electroacupuncture along with metformin lowers glucose levels and facilitates the insulin sensitivity by activating *MAPK* in steroid-induced insulin resistant rats. **(Peplow, 2015)**

Yang & Liu, 2015 observed in their study that BO's abdominal acupuncture has obvious clinical efficacy for obese type-2 diabetes mellitus, featuring in lowering the blood pressure, reducing body weight, decreasing blood glucose, improving insulin sensitivity and lowering lipid level. There was no adverse effect reported in the study. Hence, it is worthy of clinical popularization and for therapeutic application. **(Yang & Liu, 2015)**

Zhiyuan *et al.,* 2015 in their study concluded that Acu-TENS could improve the health status of the diabetes patients and can be used as a therapy in clinical application for Type 2 Diabetes Mellitus. **(Zhiyuan et al., 2015)**

Wang *et al.,* 2014 reported that the acupuncture positively regulates the glucose and lipid metabolism in the type 2 diabetes patients. Acupuncture improves the insulin resistance, enhances the body's sensitivity to insulin and improves the pancreatic islet beta-cell functions. **(Wang et al., 2014)**

3.2.9 YOGASANA, PRANAYAMA ON STRESS & TYPE 2 DM

Yoga has been the subject of research for the past few decades for the therapeutic purposes for modern epidemic diseases like mental stress, obesity, hypertension, diabetes, coronary heart diseases and chronic obstructive pulmonary disease. Individual studies report the beneficial effect of yoga in these conditions, indicating that yoga can be used as non-pharmaceutical intervention or complement to drug therapy for treatment of these conditions. **(Taneja, 2014)**

A systemic search yielded 32 articles published between 1980 and April 2007. These studies found that, yoga interventions are generally effective in reducing body weight, blood pressure, glucose level and high cholesterol. **(Yang, 2007)** It has also been postulated that yoga can rejuvenate or regenerate the β-cells of pancreas. **(Sahay & Murthy, 1988)**

Malhotra *et al.,* 2004 postulated that yoga has positive effect on general wellbeing, stresses, alertness and attentiveness without any side effects in an individual. **(Malhotra et al., 2004)**

Sahay, 2007 observed the beneficial effect of yoga in type 2 diabetes which has been attributed to increased insulin sensitivity at the target tissues which decreases insulin resistance and consequently increases the peripheral utilization of glucose. **(Sahay, 2007)**

Singh *et al.,* 2008 advocated that yoga can be considered as cost-effective and non-invasive adjuvant therapy. Apart from decreasing the dosage of oral hypoglycemic drugs or insulin, yoga can also plays vital role in delaying the progression of the disease process. **(Singh et al., 2008)**

Chaya *et al.,* 2008 reported a significant decrease in fasting plasma insulin in the yoga practitioners and is associated with increased insulin sensitivity and attenuation of negative relationship between body weight / waist circumference and insulin sensitivity. Yogasanas lead to increased sensitivity of β-cells of pancreas to the glucose signal. **(Chaya et al., 2008)**

Any chronic disease like diabetes leads to decrease in quality of life. Poor quality of life may affect compliance with treatment. A randomized controlled trial conducted by **Jyotsna *et al.,* 2012** has shown that practice of *comprehensive yogic breathing* program significantly *improves physical, psychological and social domains* and overall total quality of life. **(Jyotsna et al., 2012)**

Gopal *et al.,* 2011 from their study concluded that yoga has significant effect in ameliorating the autonomic, endocrine & psychological change brought about by the examination stress and has a beneficial effect on the immune system of individual. **(Gopal et al., 2011)**

Kauts & Sharma, 2012 conducted study with 800 adolescent students; 159 high stress students and 142 low stress students, selected on the basis of scores obtained through Stress Battery. Yoga module consisting of yoga asanas, pranayama, meditation, prayer and a value orientation programme was administered on experimental group for 7 weeks. Pre and post interventional test were conducted on both experimental and control group for their performance in concentration and memory tests. The result shows that the students who practiced yoga module yielded higher concentration levels and exhibited better short term memory. **(Kauts & Sharma, 2012)**

Madanmohan *et al.,* 2012 conducted study on the effect of yoga therapy on reation time, biochemical parameters and wellness score of peri and post-menopausal diabetic patients. They concluded that six-week yoga therapy produces significant improvement in reaction time, blood glucose and lipid profile of peri- and post-menopausal diabetes subjects. They advocated that a comprehensive yoga therapy program has the potential to enhance the beneficial effects of standard medical management of diabetes mellitus, which can be used in an effective integrative therapy program. **(Madanmohan et al., 2012)**

McDermott *et al.,* 2014 observed that, among Indians whom they are presented with elevated fasting blood glucose level, participation in an eight week yoga intervention was feasible and resulted in greater weight loss &

reduction in waist circumference when compared to control group. Yoga offers a promising lifestyle intervention for decreasing the weight-related type 2 diabetes risk factors and potentially increasing the psychological wellbeing of the individual. **(McDermott et al., 2014)**

Jyotsna *et al.,* 2014 reported that there is a significant improvement in quality of life, post prandial plasma glucose and sympathetic cardiac autonomic function tests in patients practicing Sudarshan Kriya yoga (SKY). This randomized control trial points towards the beneficial effect of yogic breathing in preventing cardiac neuropathy progression and this has important implication, as cardiac autonomic neuropathy has been considered as one of the prime factor for the sudden cardiac deaths. **(Jyotsna et al., 2014)**

Vinutha *et al.,* 2015 conducted study on 15 diabetic patients to assess the effect of Integrated approach of Yoga Therapy (IAYT) on autonomic functions. This study showed a significant improvement in autonomic functions following 1-week IAYT. They directed for further studies, needed to understand the autonomic changes following the long-term practice of yoga. **(Vinutha et al., 2015)**

Srivastava & Tiwari, 2015 investigated the effect of yoga asanas and pranayama on diabetic person. After eight weeks of practice, it was observed that asanas & pranyama reduced adults' blood sugar level, blood pressure and

glycosylated hemoglobin (HbA1c); it was found at normal level. **(Srivastava & Tiwari, 2015)**

Chimkod *et al.,* 2015 in their study demonstrated that yoga is effective in reducing the blood glucose level in patients with type 2 diabetes mellitus. **(Chimkod et al., 2015)**

A non-randomized controlled trial conducted by **Dasappa *et al.,* 2016** to study the effectiveness of yoga program in the management of diabetes, using the community health workers in the urban slums of Bangalore city revealed that yoga program was successful in improving their dietary practices, medication adherence and helpful in increasing the proportion of diabetics and hypertensive patients whom they maintain their parameters under control. **(Dasappa et al., 2016)**

A small pilot study conducted by **Mullur & Ames, 2016** reinforces the current medical evidence supporting the use of yoga combined with standard medical care to improve the health outcomes in diabetic people. **(Mullur & Ames, 2016)**

Gainey *et al.,* 2016 in their experiment of the effect of Buddhist walking meditation on glycemic control and vascular function in patients with type 2 diabetes, found after 12 weeks of practice that the maximal oxygen

consumption increased, and fasting blood glucose level decreased. Significant decrease in the level of HbA1C and systolic & diastolic blood pressure were observed in the patients practiced walking meditation. Arterial stiffness was improved and blood cortisol level was reduced. It has been concluded that Buddhist Walking meditation is superior to traditional walking program in patients with type 2 diabetes mellitus. **(Gainey et al., 2016)**

Sreedevi *et al.,* 2017 concluded that the effect of yoga and peer support on glycemic outcomes was incremental and recommended that longer term studies are necessary to ascertain the benefits shown by this feasibility study. **(Sreedevi et al., 2017)**

A meta-analysis was carried out by **Cui *et al.,* 2017** to evaluate the efficacy of yoga in adults with type 2 diabetes mellitus. The PubMed, EMBASE and Cochrane database were searched to obtain eligible randomized controlled trials with primary outcome being FBG, secondary outcome includes HbA1C, total cholesterol, HDL, LDL, Triglycerides and PPBG. In their meta-analysis, the available data evidence suggests that *yoga benefits adult patients with type 2 diabetes mellitus;* but due to limited methodology and the potential heterogeneity, further studies are needed to support the research findings and investigations were needed to find out the long-term effects of yoga in type 2 diabetes mellitus. **(Cui et al., 2017)**

3.3 INTERVENTION

'Svasa' means 'inspiratory breath' and *'Prasvasa'* means 'expiratory breath'. Pranayama is defined in Patanjali Yoga Sutra (chapter II-49) as *"Tasmin Sati Svasa Prasvasayorgativicchedah Pranayamah"* – Regulation of breath or control of Prana is the stoppage of inhalation and exhalation, which follows after securing that steadiness of posture or seat (Asana). Just as a goldsmith removes the impurities of gold by heating it in the hot furnace by strongly blowing the blow-pipe, in the practice of pranayama by blowing his/her lungs, impurities were removed from the body and Indriyas. **(Sivananda, 2000)**

3.3.1 BHASTRIKA PRANAYAMA

ऊर्वोरुपरि संस्थाप्या शुभे पादतले उभे ।

पद्मासनं भवेदेतत्सर्वपापप्रणाशनम् ।। ५९ ।।

Ūrvorupari samsthāpya śubhe pādatale ubhe |

Padmāsanam bhavedetatsarvapāpapranāśanam ||

Meaning: *Placing both soles of the feet on top of the thighs is padmasana which destroys all sins (bad karma).*

- Hatha yoga pradipika *(chapter2, verse 59)*

सम्यक्पद्मासनं बद्ध्वा समग्रीवोदरः सुधीः ।

मुखं संयम्य यत्नेन प्राणं घ्राणेन रेचयेत् ।। ६० ।।

Samyakpadmāsanam baddhvā samaghrīvodarah sudhīh |

Mukham samyamya yatnena prānam ghrānena rechayet ||

Meaning: *Sitting properly in padmasana, keeping neck and abdomen in alignment, exhale prana through the nose.*

- Hatha yoga pradipika *(chapter2, verse 60)*

यथा लगति ह्रत्कंठे कपालावधि सस्वनम् ।

वेगेन पूरयेच्चापिह्रत्पद्मावधि मारुतम् ।। ६१ ।।

Yathā laghati hrtkanthe kapālāvadhi sasvanam |

Veghena pūrayechchāpi hrtpadmāvadhi mārutam ||

Meaning: *And again the air should be quickly inhaled upto the heart lotus. Accordingly, the resounding is felt from the heart and throat up to the cranium.*

- Hatha yoga pradipika *(chapter2, verse 61)*

पुनर्विरेचयेत्तद्वत्पूरयेच्च पुनः पुनः ।

यथैव लोहकारेण भस्त्रा वेगेन चाल्यते ।। ६२ ।।

Punarvirechayettadvatpūrayechcha punah punah |

Yathaiva lohakārena bhastrā veghena chālyate ||

Meaning: *In that way it (the breath) is inhaled and exhaled repeatedly, with the same motion as a pair of bellows being pumped.*

- Hatha yoga pradipika *(chapter2, verse 62)*

तथैव स्वशरीरस्थं चालयेत्पवनं धिया ।

यदा श्रमो भवेद्देहे तदा सूर्येण पूरयेत् ।। ६३ ।।

Tathaiva svaśarīrastham chālayetpavanam dhiyā |

Yadā śramo bhaveddehe tadā sūryena pūrayet ||

Meaning: *Thus, in this way, one keeps the breath moving with mindfulness (awareness) and body steadiness. When the body is tired then inhale through the right nostril.*

- Hatha yoga pradipika *(chapter2, verse 63)*

यथोदरं भवेत्पूर्णमनिलेन तथा लघु ।

धारये न्नासिकां मध्यातर्जनीभ्यां विना द्वढम् ।। ६४ ।।

Yathodaram bhavetpūrnamanilena tathā laghu |

Dhārayennāsikām madhyātarjanībhyām vinā drdham ||

Meaning: *Accordingly, when the abdomen becomes full of air, then quickly hold the nostrils (and breath) firmly, without using the index and middle fingers (i.e. using the thumb and ring finger as in nasikagra mudra).*

- Hatha yoga pradipika *(chapter2, verse 64)*

विधिवत्कुंभकं कृत्वा रेचयेदिडयानिलम् ।

वातपित्तशलेष्महरं शरीराग्निविवर्धनम् ।। ६५ ।।

Vidhivatkumbhakam krtvā rechayedidayānillam |

Vātapittaśleshmaharam śarīrāghnivivardhanam ||

Meaning: *Having performed (pranayama and) retention systematically, exhale through the left nostril. Thereby imbalances of wind, bile and mucus are annihilated and the digestive fire increased.*

- Hatha yoga pradipika *(chapter2, verse 65)*

कुंडली बोधकं क्षिप्रं पवनं सुखदं हितम् ।

ब्रह्मनाडीमुखे संस्थकफाद्यर्गलनाशनम् ।। ६६ ।।

Kundalī bodhakam kshipram pavanam sukhadam hitam |

Brahmanādīmukhe samsthakaphādyarghalanāśanam ||

Meaning: *This (bhastrika) quickly arouses kundalini. It is pleasant and beneficial, and removes obstruction due to excess mucus accumulated at the entrance to brahma nadi.*

Hatha yoga pradipika *(chapter2, verse 66)*

सम्यग्गात्रसमुद्भूतग्रंयित्रयविभेदकम् ।

विशेषेणौव कर्तव्यं भस्त्राख्यं कुंभकंत्विदम् ।। ६७ ।।

Samyaghghātrasamudbhūtaghranthitrayavibhedakam |

Višeshenaiva kartavyam Bhastrākhyam kumbhakam tvidam ||

Meaning: *This kumbhaka called bhastrika enables the three granthis (psychic/pranic knots) to be broken. Thus it is the duty of the yogi to practice bhastrika.*

Hatha yoga pradipika *(chapter2, verse 67)*

3.3.2 KAPALBHATI PRANAYAMA

भस्त्रावल्लोहकारस्य रेचपूरौ ससंभ्रमौ ।

कपालभातिर्विख्याता कफदोषविशोषणी ।। ३५ ।।

Bhastrāvallohakārasya rechapūrau sasambhramau |

Kapālabhātirvikhyātā kaphadoshavišoshanī ||

Meaning: *Perform exhalation and inhalation rapidly like the bellows (of a blacksmith). This is called kapalbhati and it destroys all mucous disorders.*

- Hatha yoga pradipika *(chapter2, verse 35)*

षट्कर्मनिर्गतस्थौल्यकफदोषमलादिकः ।

प्राणायामं ततः कुर्यादनायासेन सिद्धयति ।। ३६ ।।

Shatkarmanirghatasthaulyakaphadoshamalādikah |

Prānāyāmam tatah kuryādanāyāsena siddhyati ||

Meaning: *By the six karmas (shatkarma) one is freed from excesses of the doshas. Then pranayama is practiced and success is achieved without strain.*

- Hatha yoga pradipika *(chapter2, verse 36)*

प्राणायामैरेव सर्वे प्रशुष्यन्ति मला इति ।

आचार्याणां तु केषांचिदन्यत्क्रम न संमतम् ।। ३७ ।।

Prānāyāmaireva sarve praśushyanti malā iti |

Āchāryānām tu keshāmchidanyatkarma na sammatam ||

Meaning: *According to some teachers, pranayama alone removes impurities and therefore they hold pranayama in esteem and not the other techniques.*

Hatha yoga pradipika *(chapter2, verse 37)*

3.3.3 RESEARCH ON BHASTRIKA & KAPALBHATI PRANAYAMA

Slow pace bhastrika pranayama (respiratory rate at 6/min) shows a strong tendency to improving the autonomic nervous system through the enhanced activation of parasympathetic nervous system. **(Pramanik et al., 2009)**

M *et al.,* 2012 reported that Bhastrika pranayama is found to be effective in decreasing blood pressure and increasing heart rate immediately. They found a significant increase in the pulmonary parameters like MEP and PEFR in their study. It suggests that the practice of pranayama helps in improvement of cardiopulmonary functions. **(M et al., 2012)**

Patil & Sawant, 2012 observed that there was significant increase in the pulmonary function reflected by increase in PFT i.e. FVC, FEV, SVC, PFER & MVV. Lung vital capacity was found increased after the practice of bhastrika pranayama. It can be stated that pranayamic exercises are beneficial for better maintenance of pulmonary functions even in healthy individuals. **(Patil & Sawant, 2012)**

Agrawal *et al.,* 2015 found in their study that during bhastrika pranayama, breath holding capacity was increased near about double of its normal duration. The rate of exchange of air in the alveoli and ventilation at the lower areas of lungs increased, which increase the level of oxygen and decreases the level of

carbon di-oxide level in the blood, which in-turn increased the breath retention duration during the practice of bhastrika pranayam. **(Agrawal et al., 2015)**

Suryawanshi *et al.,* 2015 observed a significant increase in the pulmonary function test, lung vital capacity was found to increased which implies that the practice increase the physiological strength of the lungs and is beneficial for the better maintenance of the pulmonary functions. **(Suryawanshi et al., 2015)**

Bal *et al.,* 2009 made a study on the effect of kapalbhati on Peak Expiratory Flow Rate and Pulse Rate. The study was conducted on 30 female inter-college players of yoga from Guru Nanak Dev University, Amritsar (Punjab), INDIA. Six weeks kapalabhati program showed a significant improvement in peak expiratory flow rate and pulse rate in the individual participated in the study. **(Bal et al., 2009)**

Ruhal *et al.,* 2010 in their study of effect of kapalbhati on selected body composition variables reported that after 12 weeks of practice of kapalbhati, there was significant effect on the body fat percentage, lean body mass, body water content and basal metabolic rate of the individual who practiced kapalbhati pranayama. **(Ruhal et al., 2010)**

Kekan, 2013 from his study concluded that body mass index and abdominal skinfold thickness showed a decline after the practice of kapalbhati pranayama.

So, Kapalbhati pranayama can be practiced regularly to reduce the obesity. **(Kekan, 2013)**

Dinesh *et al.,* 2013 concluded that a 12 weeks practice of kapalabhati pranyama showed improvement in the cardio-respiratory parameters with significant decrease in the RR, which may be attributed to a calm and stable mind-emotion complex. Hence, the practice of kapalabhati pranayama through the *psychosomatic mechanism enhances the health & well-being* of the individual. **(Dinesh et al., 2013)**

BAL, 2015 in his study on the impact of short-term training of kapalbhati pranayama on components of health-related fitness found that there was a significant difference was found in the components of health-related fitness (i.e., Cardiorespiratory Endurance, Flexibility, and Percentage of body fat, Fat weight & Lean Body Weight). However, there was insignificant differences were noted in muscular strength and muscular endurance of university level girls with whom the study was conducted. **(BAL, 2015)**

Jain, 2016 concluded that there was a significant improvement in cardiovascular and pulmonary parameters in pre-hypertensive obese subjects. Six weeks Kapalabhati practice normalizes the high blood pressure of pre-hypertensive obese people; therefore it may be useful tool to keep the person healthy if practiced regularly throughout the life. **(Jain, 2016)**

Bal, 2016 reported that there was a significant difference in Tidal Volume (V_T), Expiratory Reserve Volume (ERV), Vital Capacity (VC) and Inspiratory Capacity (IC) in the experimental group after 4-week training of kapalbhati pranayama. But there was insignificant difference were noted in Inspiratory Reserve Volume (IRV) of the subjects participated in the study. **(Bal, 2016)**

4.0 MATERIALS AND METHODS

4.1 STUDY SAMPLES:

A total number of fifty (50) subjects of both sexes within age group of 30 – 55 years have participated in the study. The subjects were screened through a routine medical checkup and those who satisfy diagnostic criteria for Type 2 Diabetes Mellitus were recruited for the study after getting informed consent.

4.1.1 DESCRIPTION OF POPULATION SUBJECTED FOR STUDY

The study population was selected from the Out Patients Department (OPD) of Government Yoga and Naturopathy Medical College & Hospital, Arumbakkam, Chennai. Based on the inclusion and exclusion criteria, those subjects satisfying diagnostic criteria for Type 2 Diabetes Mellitus with their willingness were subjected for the study. The population has fair representation from the rural and urban areas. The study population was from varied socioeconomic status particularly belonging to lower and middle socioeconomic groups.

4.1.2 PLACE OF STUDY

The study was conducted at Government Yoga and Naturopathy Medical College & Hospital - a teaching institute and a 50 bedded hospital situated at Chennai, catering to the needs of population from all over Tamil Nadu.

4.2 ETHICAL CONSIDERATION:

4.2.1 ETHICAL CLEARANCE

Ethical clearance for conducting the study was obtained from the Institutional Ethical Committee (IEC), after submitting the complete details of the research proposal to the committee.

4.2.2 ETHICS PERTAINING TO SAMPLE SUBJECTS

Subjects who fulfilled criteria for sample selection were explained about the purpose of study and provided with the information sheet containing details of the study viz. nature of the study, intervention used, test done for analysis of outcome of the study and rights of the sample being as research subject. The subjects have been given time and opportunity to make further clarifications.

Once the subject express their interest to take part in the study they have been explained about the importance of their role in research study and their rights to withdraw anytime from the study with prior information well in advance to the investigator.

The informed consent form with willingness details in English and Tamil language was given to the subjects. The subjects were asked to go through the consent form and they have been provided further clarifications as and when required. All the subjects expressed their willingness to participate in the study by presenting a signed informed consent form.

4.3 CRITERIA FOR SAMPLE SELECTION:

4.3.1 CRITERIA FOR DIAGNOSIS

Table 4.1 - Diagnosis of diabetes and pre-diabetes **(Pearson & McCrimmon, 2014)**

Diabetes is confirmed by either:		
• Plasma glucose in random sample or 2 hrs after a 75 g glucose load ≥ 11.1 mmol/L (200 mg/dL) ***or*** • Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL) *In asymptomatic patients, two diagnostic tests are required to confirm diabetes.*		
Pre-diabetes is classified as		
• Impaired fasting glucose = fasting plasma glucose ≥ 6.0 mmol/L (108 mg/dL) and < 7.0 mmol/L (126 mg/dL) • Impaired glucose tolerance = fasting plasma glucose < 7.0 mmol/L (126 mg/dL) and 2-hr glucose after 75g oral glucose drink 7.8-11.1 mmol/L (140-200 mg/dL)		
Oral Glucose Tolerance Test (OGTT) – Venous plasma glucose		
	Fasting	**2hrs after glucose load**
Fasting hyperglycemia	6.1-6.9 mmol/L (110-125 mg/dL)	< 7.8 mmol/L (< 140 mg/dL)
Impaired glucose tolerance	< 7.0 mmol/L (< 126 mg/dL)	7.8-11.0 mmol/L (140-199 mg/dL)
Diabetes	≥ 7.0 mmol/L (≥ 126 mg/dL)	≥ 11.1 mmol/L (≥ 200 mg/dL)
**The use of HbA1C for diagnosis is uncertain*		

4.3.2 INCLUSION CRITERIA

- Age group between 30 – 55 years
- Both gender (male and female)
- Diagnosed with type 2 diabetes mellitus.
- On conventional medical drug therapy/complementary and alternative medical drug therapy for diabetes mellitus.

4.3.3 EXCLUSION CRITERIA

- Type 2 Diabetes subjects on Insulin therapy.
- Type 1 Diabetes Mellitus (Type 1 DM)
- Gestational Diabetes Mellitus (GDM)
- Patients presents with associated complications of diabetes such as retinopathy, neuropathy, nephropathy, etc.
- Pregnancy and lactation.
- Obesity associated with back pain.
- Hypertension / Arrhythmias / Ischemic Heart Disease.
- Recent history of surgery.
- Patients with other cardio-vascular complications, respiratory complaints, hernia, vertigo, and debilitating diseases.
- Individual who already practicing yoga for a month or more.

4.4 STUDY DESIGN

A prospective pre-post study design – Subjects satisfying eligibility criteria (Inclusion & Exclusion criteria) were selected and randomized to receive their intervention (either Bhastrika / Kapalbhati Pranayama). Pre- and Post-interventional objective variable like FBS & PPBS were collected on Day 1 and Day 30 respectively.

4.4.1 SAMPLE SIZE

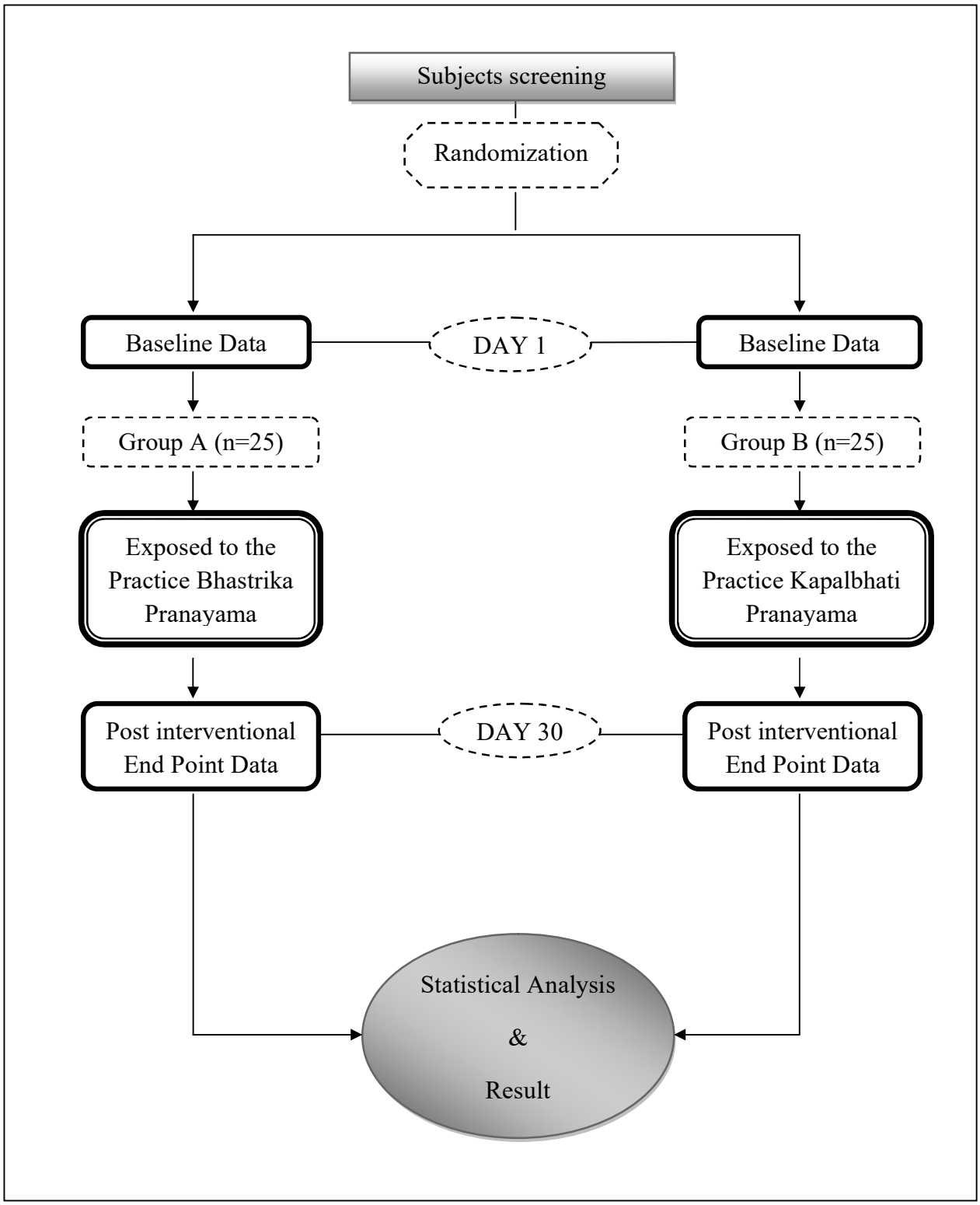
Sample size (n=50).

In both groups twenty five eligible and willing subjects i.e., Group A (n=25) and Group B (n=25), whom they showed their willingness to participate in the study by signing the informed consent, were randomized using the random numbers generated by the randomization method and then allocated for the study.

4.4.2 RANDOMIZATION AND GROUPING

Randomization was done using online team-generator tool available at the following link - https://www.randomlists.com/team-generator. Team A (Group A) was subjected to the practice of Bhastrika Pranayama and Team B (Group B) was subjected to the practice of Kapalbhati Pranayama, for 30 days respectively.

Figure 4.1 - Trial Profile



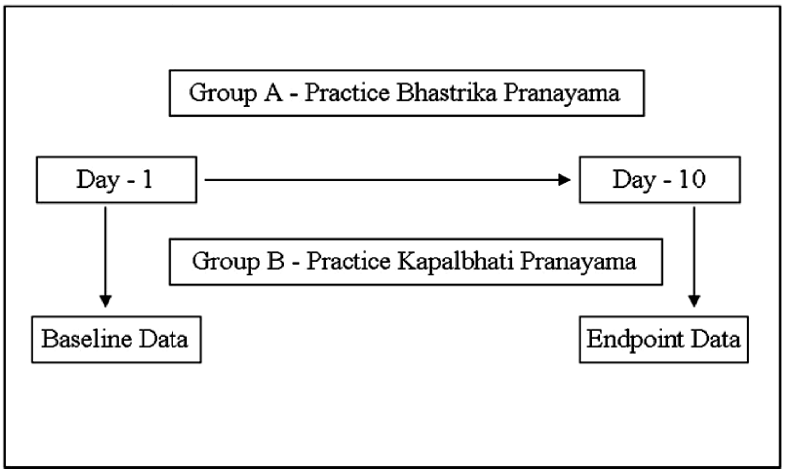
4.5 ASSESSMENT OF OBJECTIVE VARIABLE

The pre-interventional (baseline) and post-interventional (end-point) assessment of the objective variable viz. Fasting Blood Glucose (FBG) and Post-Prandial Blood Glucose (PPBG) were done.

4.5.1 ASSESSMENT INTERVAL

The pre-interventional data/baseline data is collected on day 1, before the selected subjects being subjected to the intervention and the post-interventional/end point data were collected on the last day of the study (Day 30) after completion of intervention. Both the baseline data and the end-point data were collected from subjects of both groups (Group A and Group B) separately.

Figure 4.2 - Illustration of Data Points



4.6 INTERVENTION

4.6.1 TEST INTERVENTION

The selected subjects after being allocated to the specific group were properly trained with their specific intervention i.e. Group A subjects were trained with *'Bhastrika Pranayama'* and Group B subjects were trained with *'Kapalabhati Pranayama'* for a week, before they are actually subjected into the study (Intervention Period).

4.6.1.1 PROCEDURE OF TEST INTERVENTION FOR GROUP A

Bhastrika

Sit in a comfortable meditative pose. Keep the head and spine straight. Take a slow deep breath in. Breathe out quickly and forcefully through both the nostril but do not strain, and immediately afterwards breath in with the same force. (When you breath out the abdomen comes in and the diaphragm contracts. When you breathe in the diaphragm relaxes and the abdomen moves out. These movements should be slightly exaggerated) *Keep the breath rhythmic, inhalation and exhalation must be equal*. After the last exhalation, inhale slowly and deeply through both nostril and exhale rapidly through the mouth. Perform *jalandhara bandha*, *uddiyana bandha* and *moola bandha* in this order. Hold retention as long as comfortable; on completion of retention, release moola bandha, uddiyana bandha, and then jalandhara bandha. Between each round, concentrate on the natural breath or mid-eyebrow centre. Practice five rounds of fifty breaths. **(Muktibodhananda, 2012)**

4.6.1.2 PROCEDURE OF TEST INTERVENTION FOR GROUP B

Kapalabhati

Sit in a comfortable meditative pose, preferably *siddhasana/siddha yoni asana*. Close the eyes and relax, keeping the spine erect. Place the hands in either *jnana or chin mudra*. Inhale deeply and perform fifty fast respirations through both nostrils placing more emphasis on exhalation. Inhalation should be short. After the last exhalation, inhale deeply through the nose and exhale quickly through the mouth, slightly pursing the lips. With *kumbhaka* (breath holding), perform *jalandhara bandha*, *moola bandha*, and *uddiyana bandha* in this order, but almost simultaneously. Maintain kumbhaka and the bandhas for as long as possible and count the duration. Before inhaling, release moola bandha, uddiyana bandha and jalandhara bandha in this order. When the head is raised, inhale slowly through the nose. Practice five rounds of fifty breaths. **(Muktibodhananda, 2012)**

4.7 DATA EXTRACTION & ANALYSIS

4.7.1 DATA EXTRACTION

FBG determined using the fasting blood sample and PPBG determined using 2 hours post food intake blood sample collected using a simple finger prick method and the test is done using "*ACCU-CHEK ACTIVE - BLOOD GLUCOSE MONITORING SYSTEM"*.

4.7.2 DATA MANAGEMENT

The collected baseline and end-point data of the outcome variable were managed in Microsoft Excel Sheet (Version 2007)

4.7.3 DATA ANALYSIS

The statistical analysis was done using Stats 9.0 (College, Station, Texas, USA). Data are expressed in mean ± standard deviation of mean. Paired‘t’ test were employed to analyze the intra-group outcome biochemical parameter (FBS & PPBS) and Two-sample ‘t’ test with equal variances were employed to compare inter-group means of outcome biochemical parameter (FBS & PPBS). For all the analysis, 95% confidence intervals is used and the ‘*p’* value *<0.05* is considered to be as statistically significant.

5.0 RESULT

The present study was conducted to compare and evaluate the efficacy of bhastrika and kapalbhati pranayama in the management of type 2 diabetes mellitus. The efficacies of the interventions were assessed based on the outcome variable viz. Fasting Blood Sugar (FBS) and 2-hour Post-Prandial Blood Sugar (2-hrPPBS). For all the statistical analysis 95% confidence interval is used and the '*p*' value < 0.05 is considered to be as statistically significant.

The extracted baseline and end-point data were analyzed using, paired't' test to evaluate the outcome within the group and the two-sample 't' test with equal variances to compare and evaluate the outcome of the intervention between the group. In a Paired't' test employed, the following variables of Group A, FBS *(p<0.0001) (t=39.19)*, 2-hr PPBS *(p<0.0001) (t=42.2)* and the variables of Group B, FBS *(p<0.001) (t=35.38)*, 2-hr PPBS *(p<0.001)* (t=19.46) shows a statistically significant change.

Meanwhile, in a Two-sample't' test with equal variances (difference in FBS [fbspre-fbspost] and PPBS [ppbspre-ppbspost]) employed between the groups, the following variable FBS *(p<0.0001) (t=9.69)* and 2-hr PPBS *(p<0.0001) (t=12.18)* shows a statistically significant change.

Fig 5.1 (a) - Sex distribution in Group A

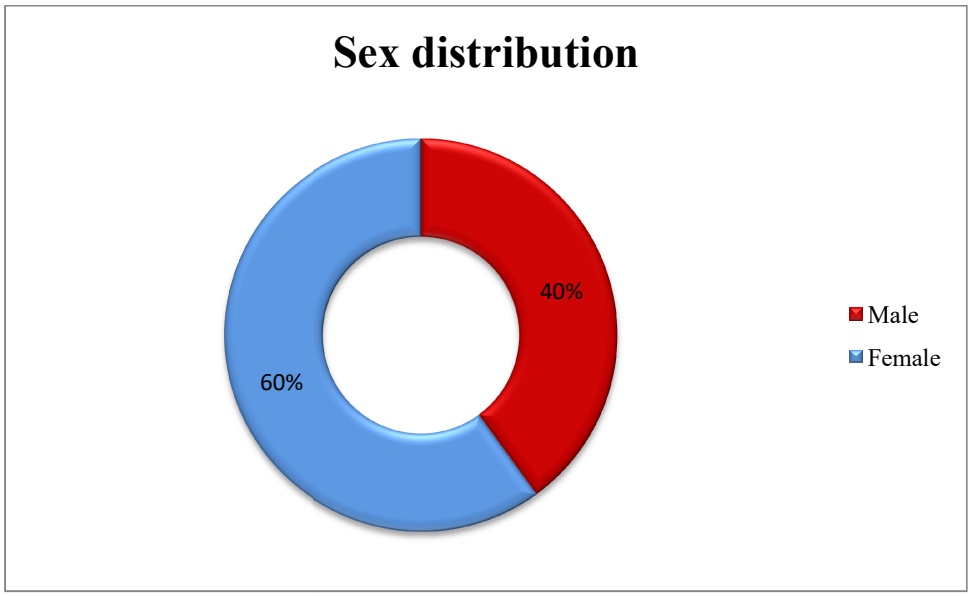


Fig 5.1 (b) - Sex distribution in Group B

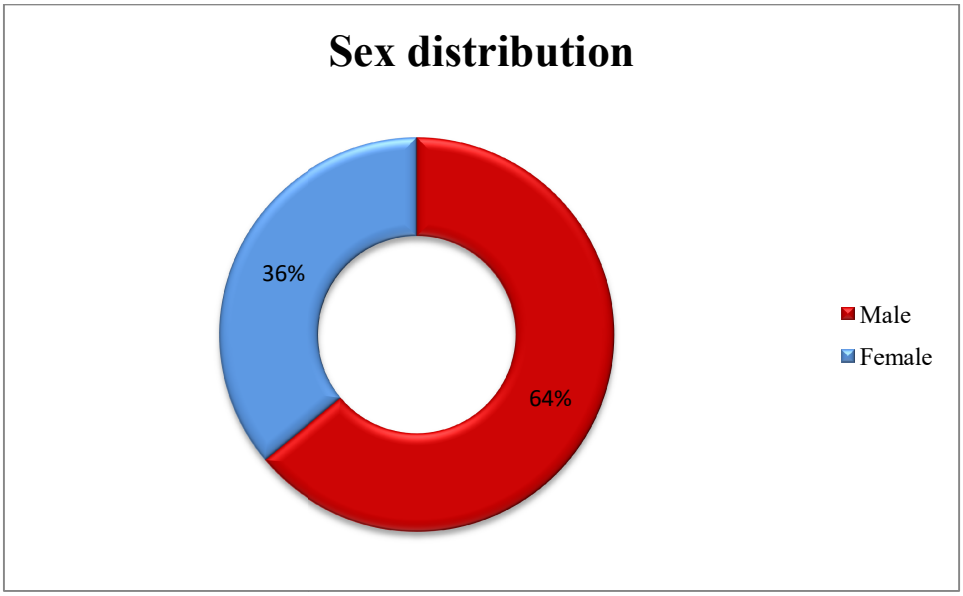


Table 5.1 – Effect of Bhastrika pranyama on Fasting Blood Sugar (FBS) and 2-hr Post-Prandial Blood Sugar (PPBS) in Type 2 Diabetes Mellitus

Variables	Group A		*p* Value
	Mean ± S.D.		
	Pre	Post	
Fasting Blood Sugar (FBS)	177.4 ± 29.57	143.32 ± 26.49	0.001
2-hr Post-Prandial Blood Sugar (PPBS)	229.52 ± 25.76	196.68 ± 25.79	0.001

Table 5.2 - Effect of Kapalbhati pranyama on Fasting Blood Sugar (FBS) and 2-hr Post-Prandial Blood Sugar (PPBS) in Type 2 Diabetes Mellitus

Variables	Group B		*p* Value
	Mean ± S.D.		
	Pre	Post	
Fasting Blood Sugar (FBS)	180.2 ± 20.56	156.72 ± 21.06	0.001
2-hr Post-Prandial Blood Sugar (PPBS)	225.36 ± 22.76	207.28 ± 24.72	0.001

Fig 5.2 – Effect of Bhastrika Pranayama on Fasting Blood Sugar

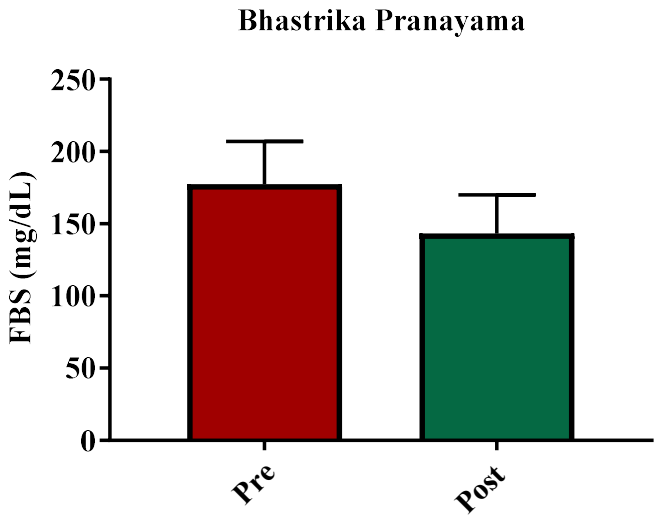


Fig 5.3 – Effect of Kapalbhati Pranayama on Fasting Blood Sugar

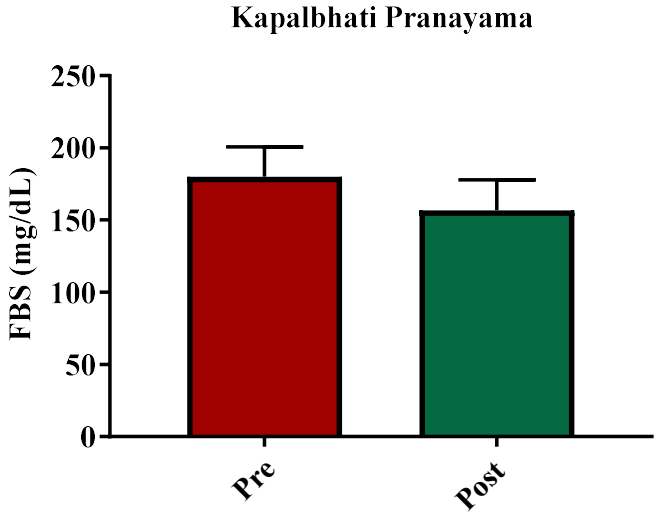


Fig 5.4 – Effect of Bhastrika Pranayama on Post-Prandial Blood Sugar

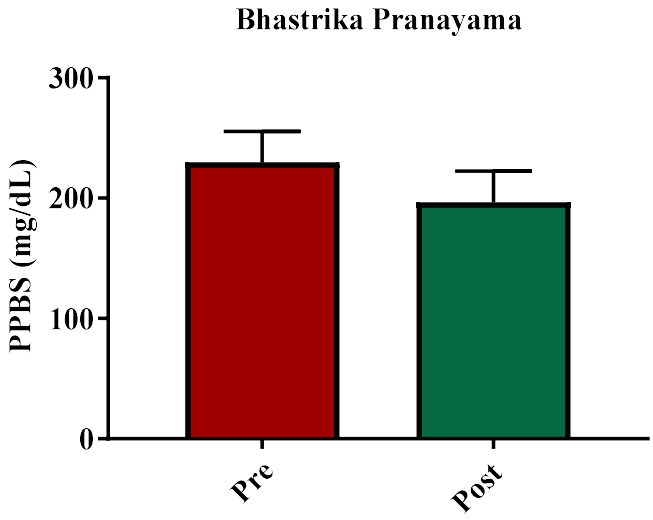


Fig 5.5 – Effect of Kapalbhati Pranayama on Post-Prandial Blood Sugar

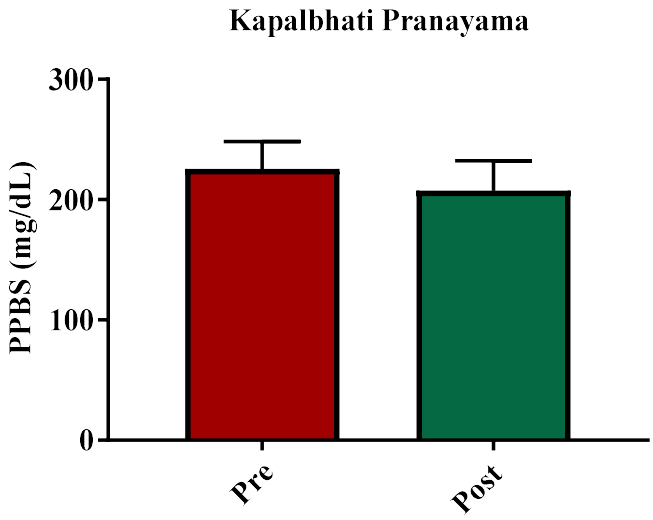


Table 5.3 – Comparison of the difference in Fasting Blood Sugar (fbspre-fbspost) and 2hr Post-Prandial Blood Sugar (ppbspre-ppbspost) between the Group A and Group B

Variables	Group	Mean ± S.D.	*p* Value
Fasting Blood Sugar (FBS)	Bhastrika	34.08 ± 4.35	0.001
	Kapalbhati	23.48 ± 3.32	
2-hr Post-Prandial Blood Sugar (PPBS)	Bhastrika	32.84 ± 3.89	0.001
	Kapalbhati	18.08 ± 4.65	

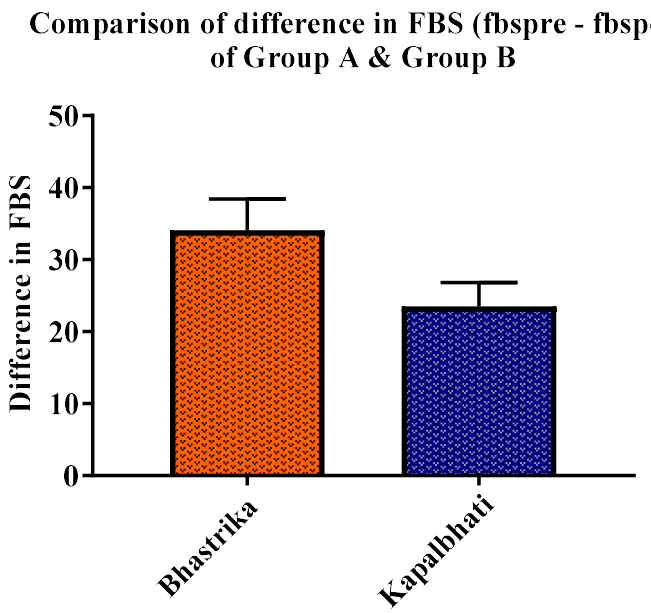

Comparison of difference in FBS (fbspre - fbspost)
of Group A & Group B
Difference in FBS
50
40
30
20
10
0
Bhastrika
Kapalbhati

Fig. 5.6

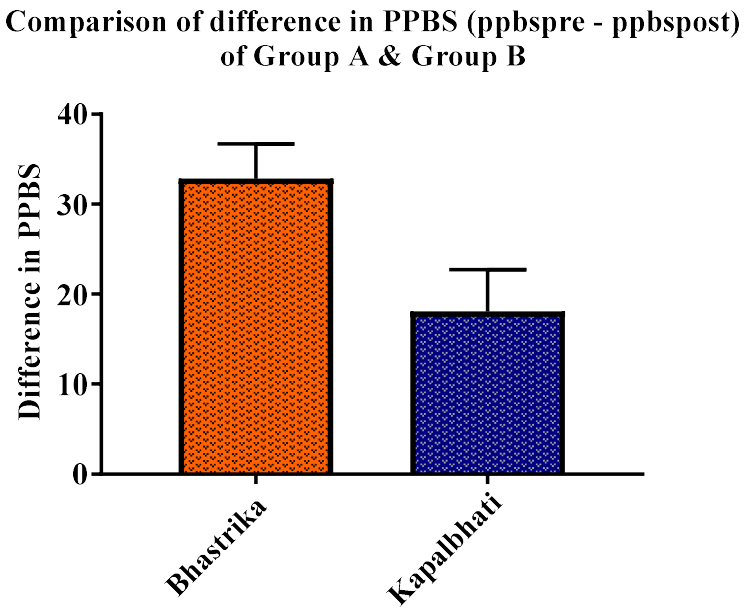

Comparison of difference in PPBS (ppbspre - ppbspost)
of Group A & Group B
Difference in PPBS
40
30
20
10
0
Bhastrika
Kapalbhati

Fig. 5.7

6.0 DISCUSSION

Psychological and physical stresses play a significant role in the development of the hyperglycemia, in the setting of type 2 diabetes mellitus. **(Batch & Surwit, 2008)** Although the human studies on the role of stress in the development and course of type 2 diabetes mellitus are few, a large number of animal studies supports that stress reliably produces hyperglycemia. Furthermore, there is a mounting evidence of the autonomic contributions to the pathophysiology of diabetes mellitus both in animals and in humans. **(Surwit et al., 1992)** Many of the modern researches and the goal of management in conventional medicine focus mainly on the various mechanism, chemical mediators and the genes which helps to promote insulin secretion / sensitize the insulin or those substance which bring down the glucose nearly normal levels. There are very few or at times no importance has been given for the psychological dimension of the patients suffering with diabetes mellitus, which has a strong influence on the hormones and endocrine functions through Psycho-neuro-endocrine-immune mechanism.

Yoga works on all aspects of the human viz. physical, vital, mental, emotional, psychic and spiritual planes. **(Saraswati, 2009)** Yoga has been shown to have effects on most physiological systems of the body. Among the various biochemical effects of yoga, a decrease in cortisol levels and control of glucose levels in diabetic subjects have been reported. **(Gopal et al., 2011)** Earlier studies in yoga have showed the importance and role of yoga in stress management, diabetes management in separate;

but there is a scanty information or study about the management of diabetes through stress control.

Pranayama has its effect not only on the breathing mechanism but also on other organs of the body. While practicing vigorous pranayama (fast breathing technique), the mind becomes inactive and tranquil for some time. By practicing retention of breath, mind becomes relaxed and there is no other way to bringing about relaxation of mind because thoughts always continuous to come in mind. **(Saraswati, 2002)**

The present study was done to evaluate and compare the outcome of the practice of Bhastrika and Kapalbhati pranayama (fast breathing technique) for five weeks in patients with type 2 diabetes mellitus. A statistically significant change was observed in the biochemical parameters (outcome variable) viz. Fasting Blood Sugar and Post-Prandial Blood Sugar in both the groups which implies both the pranayama were effective in reducing the blood glucose level (within nearly normal limits). While, comparing the difference in the fasting and post-prandial blood sugar (diff fbs = fbspre-fbspost; diff ppbs = ppbspre-ppbspost) of both the groups, Group A (Bhastrika) subjects showed a better reduction both in fasting as well as post-prandial blood sugar level compared to that of the Group B (Kapalbhati) subjects.

The observed significant changes in the biochemical parameters showed decreasing trend in mean values of pre and post FBS & PPBS of both the groups whereas when the mean of the end point data of the biochemical parameters [mean FBS (end point)

of Group A & mean of the FBS (end point), mean PPBS (end point) of Group A & mean PPBS (end point) of Group B] are subjected to two-sample 't' test, the statistical significant change was not observed probably due to less sample size and less duration of intervention. Hence longer duration of the study may be conducted with more number of the samples to observe the outcome.

A literature review was made by *Singh VP et al, 2005* to understand interrelated psycho-Neuro-Endocrine and Immunological mechanism of action of yoga in Type 2 Diabetes Mellitus. The published literature concerning mechanisms of action of Yoga in Type 2 diabetes mellitus emphasizing psycho-neuro-endocrine or immunological relations was retrieved from Pubmed. The postulated mechanism of action of yoga is through *parasympathetic activation and anti-stress mechanism*. Yoga reduces the perceived stress and *hypothalamic-pituitary-adrenal axis (HPA axis) activation,* thereby improving the overall metabolic and psychological profiles, viz. increasing insulin sensitivity, improving glucose tolerance and lipid metabolism. **(Singh et al., 2015)**

Pal et al., 2004 conducted a study to find out the effect of short-term practice (3 months) of breathing exercise on autonomic functions in normal healthy volunteers. A total of 60 male undergraduate students were randomly divided into two groups: slow breathing group and fast breathing group. They observed that there was increased parasympathetic activity and decreased sympathetic activity in slow breathing group whereas no significant change in group practiced fast breathing. **(Pal et al., 2004)**

In a study conducted by *Jyotsna et al., 2013* randomized 120 patients either to standard diabetic care or to a yogic breathing program for 6 months. At the end of the study, quality of life and post prandial plasma glucose level have significantly improved in the group practicing yoga, compared with baseline value, but there was no significant improvement in fasting plasma glucose level and HbA1c. **(Jyotsna et al., 2013)** While, our present study has showed the improvement both in fasting and post-prandial blood glucose level in both group of subjects (Group A & Group B) practiced fast breathing technique.

In a previous study conducted by *Thangavel et al., 2014* to evaluate the effect of 12 weeks training of slow and fast pranayama on handgrip strength and endurance in young healthy volunteers of JIPMER population, they observed a significant change only in hand grip endurance (HE) in slow pranyama group whereas significant improvement in hand grip strength (HGS) as well as endurance (HGE) in fast pranayama group. They concluded that pranayama training decreases sympathetic activity resulting in mental relaxation and decreased autonomic arousal, and thereby decreasing force fluctuation during isometric concentration. **(Thangavel et al., 2014)**

In another interesting study conducted by *Shende et al., 2013* short-term intervention of pranayama on blood glucose level in medical students, they noticed a significant reduction in stress in medical students which in turn improved both fasting and post meal blood glucose level. **(Shende et al., 2013)** Equivalently our present study also

demonstrated a significant reduction in both fasting and post prandial blood glucose level in diabetic subjects of both groups (Group A & Group B).

Altogether, the practice of Bhastrika as well as Kapalbhati pranayama among type 2 diabetic subjects showed, a significant reduction in fasting- as well as post-prandial blood sugar level which might have been resulted due to a *decreased sympathetic activity and improved parasympathethic activity* via *tranquil effect and stress curtailment induced by the pranayama practice* as postulated in the previous research studies. Further, from the difference in the fasting and post-prandial blood sugar level of group A and group B, it is inferred that the practice of bhastrika pranayama is more effective in reducing the blood sugar level than kapalbhati pranayama in a short-term practice. However, the study of longer duration, with large number of sample is necessary to reaffirm the assertion.

6.1 STRENGTH OF STUDY

The following were the strength of our present study:

- Effective randomization
- Equally distributed group of subjects
- No dropouts in study
- Significant improvement in outcome variables
- No adverse reaction occurred during study

6.2 LIMITATION AND DRAWBACKS

- Smaller sample size
- Short duration of intervention
- Stress tool is not used for study
- Blinding was not possible
- Subjects were not on strict and uniform diet plan
- Other naturopathy treatments might have had its influence on the outcome variable

6.3 FUTURE PROSPECTS

- A study with larger sample, longer duration with stress tool may be needed to substantiate the results of the present study
- Further study at the molecular level may be done to study the effect of pranayama on the neuro-endocrine-immune mechanism.

7.0 CONCLUSION

The present study demonstrated that 5 weeks practice of both bhastrika as well as kapalbhati pranayama is efficacious in reducing the fasting as well as post-prandial blood sugar level in type 2 diabetic subjects. From the difference in fasting and post-prandial blood sugar of both groups, it is inferred that the practice of bhastrika pranayama is more effective than kapalbhati pranayama in reducing the blood sugar level in a short-term practice which might have been the resultant effect of decreased sympathetic activity, increased parasympathetic activity and improved insulin sensitivity in patients with type 2 diabetes mellitus, as postulated in earlier studies. However, the study of longer duration with large number of sample with stress as tool is necessary to reaffirm the assertion.

8.0 SUMMARY

Diabetes is no longer a disease of rich nation; rather it is on rise everywhere most predominantly in low- and middle-income countries. It is difficult to define or measure the role of stress in the etiology of diabetes, but there is significant evidence suggest of stress's' metabolic consequences in an individual who already suffering from chronic disease like diabetes mellitus. Yoga is a mind-body technique being holistic in nature; it is the best means for achieving physical, mental, social and spiritual well being. Pranayama (breathing exercises) appear to alter autonomic, responses to breathe holding.

The present study was done to evaluate the effect of Bhastrika and Kapalbhati Pranayama on blood glucose level in subjects with type 2 diabetes mellitus. Fifty type 2 diabetes subjects whom they were on either conventional medical therapy or herbal medical therapy were screened and recruited from the out-patients department (OPD) of Government Yoga and Naturopathy Medical College & Hospital, Chennai. The recruited subjects were randomized using computerized random numbers, thus resulting in (n=25) Group A and (n=25) in Group B. Group A was subjected to the practice of Bhastrika Pranayama and Group B was subjected to the practice of Kapalbhati Pranayama for the period of five weeks.

The assessment variables include pre- and post-interventional fasting blood glucose and post-prandial blood glucose level. Baseline data was collected on Day 1 before subjecting the selected subjects in to the intervention and the end-point data was collected on the day 30 i.e., on the last day of the intervention after the subject finishes their concerned intervention. The collected data were properly maintained for the statistical analysis.

Statistical Analysis was done with Stats 9.0 (College, Station, Texas, USA). Paired‘t’ test were employed to analyze the intra-group outcome biochemical parameter Fasting Blood Sugar (FBS) and Post-Prandial Blood Sugar (PPBS) and Two-sample ‘t’ test with equal variances were employed to compare inter-group means of outcome biochemical parameter (FBS & PPBS). The statistical analysis has shown a significant change in the biochemical parameters of the subjects of both the group (Group A & Group B). Hence, this study has shown that the short-term practice of Bhastrika as well as Kapalbhati Pranayama were potent in decreasing the fasting- and post-prandial blood sugar level in type 2 diabetes mellitus patients possibly by increasing the vagal tone and decreasing the sympathetic discharge, wherein enhances the parasympathetic activity which positively influence stress, insulin resistance, obesity and sedentary lifestyle, which are the risk factors of diabetes mellitus.

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Annexure 1: PROFORMA

Name: Age:

Gender: Male / Female Marital Status:

Religion: Occupation:

Address:

Emergency Contact: Primary Language(s):

Complaints:

History of present Illness:

Previous Illness:

Personal History:

Diet: Appetite: Digestion:

Sleep: Micturition: Bowel:

Coffee/Tea: with/without sugar Addiction:

Family History:

Treatment History:

History of Allergy to any specific drugs/food, if any:

Obstetrics & Gynecology history:

Vital data:

Height:	cms	Weight:	kg
Pulse:	beats/min	Blood Pressure:	mm/Hg
BMI:		Waist Hip ratio:	
Built:		Temperature:	

GENERAL PHYSICAL EXAMINATION:

SYSTEMIC EXAMINATION:

Cardiovascular System:

Respiratory System:

Abdomen:

Nervous System:

Endocrine System:

Genitourinary System:

Locomotor System:

Investigation:

Annexure 2: INFORMATION SHEET

We are conducting a study ***"Evaluation of the effect of Bhastrika and Kapalbhati Pranayama on blood glucose level in Type 2 Diabetes Mellitus"*** at Government Yoga and Naturopathy Medical College and Hospital, Chennai – 106. The purpose of this study is to evaluate the effect of pranayama (Yogic Breathing Exercise) on blood glucose level in Type 2 Diabetes Mellitus, which may be of use in better management of Diabetes Mellitus in future.

We need your participation in this study. Here we are assessing the changes in the blood glucose level by measuring the pre- and post interventional, Fasting and 2-hr Post-Prandial blood glucose levels.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefit to which you are otherwise entitled. The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date:

Annexure 3: INFORMED CONSENT FORM

Title of the study: Evaluation of the effect of Bhastrika and Kapalbhati Pranayama on blood glucose level in Type 2 Diabetes Mellitus

Name of the Participant:

Name of the Principal Investigator: Dr. Varun. V

Name of the Institution: Department of Yoga,

Government Yoga and Naturopathy Medical College and Hospital,

Chennai - 106

Documentation of the informed consent

I _____ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in *"Evaluation of the effect of Bhastrika and Kapalbhati Pranayama on blood glucose level in Type 2 Diabetes Mellitus"*

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.

5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.
7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.
8. I have not participated in any research study within the past _____month(s).
9. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
10. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
11. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
12. I have understood that my identity will be kept confidential if my data are publicly presented.
13. I have had my questions answered to my satisfaction.
14. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this

document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____ Signature_____

Date_____

Name and Signature of impartial witness (required for illiterate patients):

Name _____ Signature_____

Date_____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature_____

Date_____

Annexure 4: INFORMATION TO PARTICIPANTS

Investigator: Dr. Varun. V

Name of Participant:

Title: Evaluation of the effect of Bhastrika and Kapalbhati Pranayama on blood glucose level in Type 2 Diabetes Mellitus

You are invited to take part in this research/ study /procedures. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns. You are being asked to participate in this study being conducted in department of Yoga, Government Yoga and Naturopathy Medical College and Hospital, Chennai – 600 106

What is the Purpose of the Research?

To evaluate and compare the effects of Bhastrika and Kapalbhati Pranayama (Yogic Breathing Exercise) on blood glucose level in Type 2 Diabetes Mellitus

The Study Design

Fifty, Type 2 Diabetes Mellitus patients of age group between 30-55 yrs will participate in the study.

Study Procedures

The study involves assessment of pre- and post interventional, Fasting & 2-hr Post-Prandial blood glucose level. The test involves simple finger prick blood glucose analysis.

You will be required to visit the hospital during the study. You may have to come to the hospital (study site) for examination and investigations apart from your scheduled visits, if required.

Possible Risks to you

Nil

Possible benefits to you

Blood glucose level were tested free of cost thereby proper intervention can be implemented.

Possible benefits to other people

The result of the research may provide benefits to the society in terms of advancement of medical knowledge and/or therapeutic benefits to future patients.

Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research

team investigators, other study personnel, sponsors, IEC and any person or agency required by law like the Drug Controller General of India to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?

Your decisions to not to participate in this research study will not affect your medical care or your relationship with investigator or the institution. Your doctor will still take care of you and you will not lose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during course of the study without giving any reasons.

However, it is advisable that you talk to the research team prior to stopping the treatment